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# OCCUPATIONAL MEDICINE
# AND
# INDUSTRIAL HYGIENE

# OCCUPATIONAL MEDICINE AND INDUSTRIAL HYGIENE

By

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"How easily, how secretly, how safely in books do we make bare without shame the poverty of human ignorance! These are the masters that instruct us without rod or ferrule, without words of anger, without payment of money or clothing. Should ye approach them, they are not asleep; if ye seek to question them, they do not hide themselves; should ye err, they do not chide; and should ye show ignorance, they know not how to laugh. O Books! ye alone are free and liberal. Ye give to all that seek, and set free all that serve you zealously."

—Richard de Bury.

# PREFACE

Unlike the words which preface most books, these few constitute the epilogue rather than the prologue. They are written in retrospection of a completed task and at its end the author finds himself asking, *cui bono?* Will this book be looked upon as a collection of cold medical data or will he who reads it sense in it the poetry and romance of occupational medicine; will he recognize the story of great achievements, and the challenge to come into the arena where the fight is still on to make the life of those who toil worth living?

Will this book be able to define for the student, for the medical educator, and for a considerably large segment of the medical profession the true meaning of occupational medicine? Will the following pages be able to convey to the reader that occupational medicine is that phase of practice which is concerned with the working and living habits of the worker as well as with the physical conditions under which he works and lives? Will it be apparent to the medical profession that when the doctor offers and industry accepts an adequate industrial health program, the great mass of American citizens will never tolerate the political administration and control of their health? These and other questions crowd the mind as the ink dries on the last page.

Although occupational medicine has been designated in recent years as a specialty, it is by no means a restricted specialism. If properly practiced, it approaches a return to the intimate concern of a physician for his patient, so nobly exemplified by the old family doctor. In this respect occupational medicine stands in sharp contrast to the limited specialties in medicine. For occupational medicine is not interested in the patient's eyes alone, or his allergy, or his heart. It is interested in man and *all* that aids or hinders his welfare. In the rise of preventive medicine, occupational medicine has assumed the foremost role.

For too many years it was believed that it is the purpose of preventive medicine to eliminate diseases. But occupational medicine recognizes that man's health and happiness are affected by many other factors such as nutrition, mental hygiene, housing, sanitation, recreation, noise, vibration, lighting, ventilation, atmospheric pressure, climatic changes, and a myriad of other influences. To the untutored, many of these subjects would appear to belong in the field of engineering, but such an assumption is far from the truth. Any engineering plans designed to benefit mankind must take into consideration the nature of the human organism. Likewise the physician needs to know more about engineering principles and control measures before he assumes the guidance of men's lives toward comfort and health. Surely no other phase of medicine undertakes such a gamut of activities as does occupational medicine.

In thumbing through the copy of this book, the original pages having been sent to the publisher, we realize that there have been certain omissions. Critics will wonder why the subjects of nutrition, or aviation medicine, or industrial nursing, to mention a few, have not been discussed. The answer is that these problems warrant textbooks of their own. To offset any remorse in this regard the author is comforted by the fact that he has adequately combined the clinical aspects of the occupational diseases with a description of industrial processes, their appraisal and control. He feels that within these pages will be found not only his personal experience, but also material which the average physician will seek in vain in his textbooks.

To whom am I indebted? To too many individuals who must remain nameless. I am forever grateful to the pioneers in this field, the magnitude of whose labor I cannot forget. Because of their work, occupational medicine runs in larger molds and turns out finer castings. Many of these have been pioneers in this field and are still active leaders; not a few of them are fellow members of the Ramazzini Society of America. To W. B. Saunders Company I am indebted for permission to use certain materials extracted from a prior book of mine, and to the entire staff of The C. V. Mosby Company for their excellent cooperation in this present publication.

Then there are those more intimate associates. I am grateful to Lee Stockwell, my faithful secretary of ten years, who did double duty while this book was being written; to Susan Engreen, for her meticulous editing and preparing of this manuscript; and finally to a most tolerant wife who accepted graciously the impositions that were involved in her husband's proclivities for writing.

Nor should the reader be ignored in this acknowledgement. The usefulness of what has been written in these pages is dependent upon him. His application of the principles outlined in this book should further the objective to which it is dedicated.

"The knowledge which a man can use is the only real knowledge, the only knowledge which has life and growth in it and converts itself into practical power. The rest hangs like dust about the brain or dries like rain drops off the stones."

RUTHERFORD T. JOHNSTONE

Los Angeles, Calif.

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# PART ONE

## CHAPTER I

## TOMORROW'S DOCTORS

### (The Background and the Future of Industrial Medicine)

Suddenly, medicine has awakened to a fresh responsibility. In the dawn of a new day it sets forth along an untried highway into the future. In centuries past, medical science has been applied to the prevention or treatment of disease and to the relief of those who are ill. But the sorrows or troubles which arise from causes other than disease have received far too little attention. With man's emotions, with his need for happiness, for laughter, and for spaciousness, with the importance of comfort or the significance of a friendly helping hand it has rarely concerned itself.

Out of a dilemma created by traditionalism, medical science is now faced for the first time with its social and economic obligations. This sudden confrontation was brought about by World War II, when it became apparent that man's industrial productivity is strictly related to his mental and physical well-being. This discovery was not made by medical men alone but was rather the joint observation by representatives of industrial management as well as medicine.

During the war years, industrial medicine was expanded to what was widely considered a fantastic if not a fanatic scope. Certainly not all the phases of a comprehensive program of industrial medicine can be considered as medicine in the traditional sense of the word. In the war-born expansion of this program industrial medicine emerged from its lifeless past, in which it had seemed to revolve in idle circles, to become a new force in the health of the American people. Today, industrial medicine has assumed a prominence equal to any other phase of medicine, and a broader type of medical practice can be expected to develop under its influence. But before scanning future horizons, let us look at the early history of industrial medicine.

Contrary to common belief, industrial medicine is not young, for it had its inception in antiquity. To indicate its age, it is sufficient to mention only a few of the physicians in olden times who took cognizance of industrial diseases: Hippocrates described lead colic as well as the toxic properties of this metal. Galen cited diseases peculiar to miners, tanners, fullers, chemists, and others. In the *Sallier Papyri* one may read of the effects of certain occupations upon the Egyptians of an early day. Even a form of industrial hygiene was practiced by the ancients, for Pliny the Elder writes that in certain dusty trades the workmen tied bladders over their mouths to prevent the inhalation of dust. Many of the earlier physicians, such as Agricola, Martial, Celsus, Dioscorides Pedanius, and Herodicus, have discussed the influence of their occupation upon the health of workers. But intriguing and romantic though its history may be, these pages are too limited to people them with all the important personages in the field of occupational medicine who have preceded our own generation.

One name, however, cannot be passed over lightly, for it is more brilliant than all the others. In a century which gave us Newton, Galileo, Boyle, and their famous contemporaries Harvey, Malpighi, and van Leeuwenhoek, there was born in Capri, in 1633, the patron saint of industrial medicine, Bernardino Ramazzini. His ability to observe, investigate, and then to describe with a facile pen has been matched but rarely since his death in the year 1714.

After being graduated from Parma in 1659, Ramazzini continued his studies at Rome under Professor Antonio Maria Rossi. Following this, he spent a short time practicing in Viterbo but contracted malaria, which induced him to return to his native Capri to regain his health. In 1670 he moved to Modena, where he was appointed professor of medicine in the recently restored university of that city. For a few years his life was pleasant, but then there followed a succession of unfortunate clashes which precipitated a bitter struggle with his fellow practitioners. This discord gave rise to a vitriolic correspondence with his hostile confreres, but Ramazzini won this joust with his penetrating mind and easy pen.

In reviewing the life of this man, one is amazed at the diversity of his interests, his immense capacity for work, and the variety of contributions which he made to medical literature. Of him Garrison[1] states: "He was a versatile polyhistorian, poet, philosopher, erudite clinician, epidemiologist, medical meteorologist and sanitarian." An enthusiastic modern admirer of Ramazzini is Robert T. Legge, of the University of California at Berkeley. Not content with an occasional collector's item, Dr. Legge has gone to Italy to tred the same ground, live in the same towns, and delve into the little old book shops to uncover some precious bits of Ramazzini lore. Writing of the recognition accorded Ramazzini, Legge[2] states: "Distinguished men of science and letters found their way to the ducal palace (Modena). Among these was the famous Baron Gottfried Wilhelm von Leibnitz, the German philosopher who paid many visits to Ramazzini, whom he recognized as a scholar and investigator. They discussed barometic and hydrostatic problems, a subject Ramazzini studied and experimented with in relation to epidemics. Leibnitz was so impressed with his learning and publications, it is inferred, that he recommended

Ramazzini to the president of the Viennese Academy of the Curiosi Naturae to be elected to its membership with the title of Hippocrates III."

Other honors came to Ramazzini. Because of his learned dissertation on "febris tertiana" he was elected a member of the Academie of Caesareo-Leopoldina at Vienna. He was invited to lecture at the University of Padua, where he made several noteworthy addresses. During his twenty-nine years at Modena he wrote a great number of treatises and books on a wide variety of subjects, but only one of these will be mentioned at this time; namely, *De Morbis Artificum Diatriba (The Diseases of Workmen).*

Since Ramazzini was such a brilliant clinician and was so deeply concerned with a variety of diseases which were also of interest to other learned physicians of his day, one is forced to speculate as to what caused him to spend so much time in the study of the diseases of workmen. In that delightfully whimsical book called *A Blind Hog's Acorns,* McCord[3] suggests that during the years that Ramazzini was smarting under the barbs of his confreres he spent many hours among the more sympathetic craftsmen of his community. It was then that he became interested in the occupation of well diggers. Not content with talking to these workers in their "off-work" hours, he would himself descend into the wells, equipped with thermometer and barometer, to talk with the diggers, inquiring as to their health and what effect underground work had on them; he made observations on subterranean waters and on the characteristics of the antiquity in the different layers of the earth, their odors and gases. He also acquainted himself with the trades of silk workers, painters, dyers, glass makers, tanners, bakers, stone workers, metal workers, chemists, and others. All told, his famous book contains forty chapters dealing with the various diseases of working men.

It is unfortunate that a selection of the classical descriptions which Ramazzini left for posterity cannot be reproduced in this chapter. Suffice it to state that there are many examples relating the signs and symptoms of such diseases as mercury poisoning or lead poisoning which are worthy of any modern textbook on the subject. For instance, of those who work with mercury, he says: "They become asthmatic, paralytic, and liable to vertigo, their complexion assuming a dangerous ghostly aspect . . . Few such workmen continue in that way to old age, for if they do not die while young they become miserable, their neck and hands tremble, their teeth fall out and their legs are weak and maul'd with scurvy."

In addition to his classical description of the occupational diseases of his day, Ramazzini left two statements which bear repeating, not as simple observations but rather as dictums worthy of adherence by all who would practice this form of medicine. The first of these was his exhortation to *all physicians* to ask of their patients not only the usual questions regarding their health but also to inquire without fail *"of what trade are you?"* The other command of the old master was that physicians should learn the nature of the occupational diseases in shops, mills, mines, or wherever men toil. Osler believed that clinical medicine is best learned in the wards of hospitals, but Ramazzini taught that occupational diseases are most effectively studied in the actual environment of the workers.

If organized labor has ever paid tribute to Ramazzini, it has escaped this writer's attention; yet, though workers may not realize it, this man who died nearly three hundred years ago was one of their benefactors. While others may have championed the cause for better pay, shorter working hours, or better industrial relations, our patron saint dedicated his life to the improvement of the conditions under which men must toil. Upon the foundation which he established still rest the industrial hygiene and sanitation of our times. He condemned lack of ventilation and unsuitable temperatures and urged that men in dusty trades should work in spacious rooms; he counseled rest intervals in work of prolonged duration, as well as exercise and correct posture, and deplored the poor facilities for bathing which prevailed in his days.

It is said that most famous men are not appreciated until some years after their death. With Ramazzini, the reverse is true, for he became famous and was admired in his own life time, only to be almost forgotten during the succeeding decades. Recently, however, his memory, his deeds, and his books have been revived by the founding of the Ramazzini Society in America, a group of thirty-one devotees of industrial medicine. This society, of which the author is a proud member, has no officers, no dues, and no bylaws and does not even hold scheduled scientific congresses. It merely proposes to perpetuate the memory of Bernardino Ramazzini by gathering annually to exchange ideas and to toast his memory with posca or with a sweeter facsimile thereof. And, furthermore, it is the unwritten purpose of this group to stimulate the practice of industrial medicine in the United States.

But to return to the historical sketch of industrial medicine: In the eighteenth century Percival Potts became interested in the cancer of chimney sweeps, while Willan described the skin lesions in bakers. Thackrah, living in the manufacturing district of Leeds, wrote about his observations concerning the diseases of workmen. But it was the effort of Thomas Percival, a Manchester physician, which led to the first Factory Act in England. In Germany, Tissot was concerned with the hazards of stone masons, while phosphorus, the dusts, and toxic gases were also mentioned in the German literature. Following the industrial revolution, the English Factory Act of 1833 stimulated similar movements in Germany and France. It was then that the *De Morbis Artificum Diatriba* was republished and widely distributed, urging the people to enact better legislation.

In the twentieth century in England, Sir Thomas Oliver published *Dangerous Trades,* followed by *Diseases of Occupation* (1908). In collaboration with Sir Kenneth Goadby, Sir Thomas Legge wrote *Lead Poisoning and Lead Absorption.* About the same time, phosphorus poisoning received the attention of Andrews in the United States. Also in this country, Kober and Hanson as well as Kober and Hayhurst added important contributions to the literature dealing with industrial diseases. Admitting the omission of many other important names in the field of industrial diseases, we come to the present generation, but discretion forbids the author to evaluate his own contemporaries. It will remain for history to pay tribute to those who, by their investigations and the promotion of industrial hygiene, have made the United States the most progressive country in the world for the protection of the working man.

But it would be an inexcusable omission indeed not to honor the most famous daughter of Ramazzini, Dr. Alice Hamilton who, in the early years of this century, climbed the dilapidated steps of Chicago slums to visit men stricken with lead poisoning. In her early medical days she became associated with Jane Addams of Hull House fame and was most zealous in the cause of the underprivileged. It was probably for this high, humanitarian purpose which she steadfastly maintained throughout her active years that she entered the field of industrial medicine. She travelled the breadth of this land to investigate different types of occupational exposure; she wrote many articles and is the author of several noteworthy books. By her works she deserves a place among the great physicians who have influenced the course of American medicine. The author wishes to acknowledge the great pioneer work of Dr. Alice Hamilton and bows reverently in the direction of Hadlyme, where she is enjoying her sunset years.

If industrial medicine is of such ancient origin, the question must be asked what may have retarded its progress to a status comparable to that of other branches of medicine. There are those who would answer that in the United States industrial medicine in its earlier years fell into evil ways: that it became "contract practice," "insurance medicine," or "finger wrapping." To a certain degree this is true. Too often industrial medicine was only a stopgap for those whose interest lay in more lucrative fields. And yet, all these explanations are correct only in a superficial sense.

For the real answer one must go deep into the socioeconomic history of the United States and begin by asking questions of a different nature, as, for instance: Why is it that America has no adequate system of rural medicine? Why has this nation lagged in its nutritional program, and in slum clearance? In the correct answer to these questions will be found the reason why industrial medicine has been late in reaching the prominent position which corresponds to its great importance. Only within our own time has this country approached its social responsibility. Except for the occasional lone voice, our social and economic philosophy has ignored mass welfare. Industrial management was no more interested in the working environment of its employees than it was in their living conditions outside the plant. How then was it possible for industrial medicine to grow?

Organized medicine is likewise to blame. Far too long it has frowned upon this type of practice, or at least has lent it no encouragement. Those who controlled the policies of our medical societies believed that industrial medicine threatened to undermine the cherished patient-physician relationship. Yet, to no appreciable degree has that relationship ever been disturbed by industrial medicine. What the leaders of organized medicine did not realize until recently was the necessity of a program to provide medical care for the masses; they did not consider that tuberculosis, venereal disease, malnutrition, cancer, and the like are group problems which can best be prevented by utilizing the machinery which controls and motivates large groups. Until lately it has not been understood that an industrial organization is a social structure which lends itself to the application of our ability to guide human relations and our plans for mass preventive medicine. This was demonstrated by the high degree of efficiency attained by the industrial tuberculosis program[4] of World War II, through which a greater number of early cases of tuberculosis were detected than through any community-sponsored program.

Likewise, medical education has placed little emphasis on industrial medicine. A few medical schools have recently augmented their courses by adding varying degrees of instruction in this field which, however, are mostly didactic. But proper emphasis on the subject is still lacking. Under these circumstances it cannot be expected that students will be seized with a zeal to enter the field of industrial health; and even if they did so, it still remains doubtful if they will be equipped with sufficient knowledge, as long as industrial medicine is taught in a half-hearted manner by inexperienced teachers when indeed the teaching of this subject is imposed upon them while they are engaged in lecturing on subjects largely unrelated to industrial medicine.

So much for the past and its obstacles. But what of the future? Its possibilities are limitless and unpredictable. Obviously, the future will include an ever-expanding program of preventive medicine, encompassing the control or elimination of tuberculosis, cancer, heart disease, venereal disease, and other scourges; the future will continue to improve preventive measures against industrial injury or occupational illness; it will expand research in the field of industrial toxicology. But it proposes to do more—much more.

Industrial medicine contends that we must cease looking upon the working man as an eight-hour a day problem, in which only his occupational environment is of concern. No longer can the industrial laborer be classified as a mechanical component of the machinery of industry. We have realized that the worker is a member of society, whose working, playing, and living is a twenty-four-hour problem. Such a concept entails consideration of all his faculties, his capacities as well as his deficiencies. It includes a program which will safeguard his hearing, his vision, his nutrition, his skills, aptitudes, experience, intelligence, and emotions. It is the object of industrial medicine to utilize the cardiac, the epileptic, the cripple, the blind, the elderly, and all those who desire to work instead of being forced to accept the beneficence perpetrated upon them by an unwitting government. Man's work is related to his home, his social environment, his philosophy, his economics, and his happiness. Adjustment of these relationships in the largest segment of our adult population can be achieved by utilizing the facilities which industrial medicine has to offer.

No doubt, the uninformed critic will say that these aims belong in the field of social science and that they should be left to the social worker. But such a contention must be emphatically contradicted. The responsibility for the well-being of the industrial worker is the obligation of trained physicians rather than that of a group equipped only with a smattering of the physiology and psychology of man. Social agencies have not been sufficiently effective, nor have they made the most of their opportunities. The improvement of working conditions for the underprivileged is not to be left solely to legislative acts, much as these are necessary to insure full success. Politicians have not added in any measurable way to the sorely needed comfort of these great masses of people. The wage level may have been raised and the worker may have won rights which had previously been denied to him. But none of these steps can decisively improve the standard of health among the working population without the leadership of the medical profession.

It was the bitter cry of Isaiah that with the multiplication of the nations, their joys had not been increased. True it is, that much sorrow and strife will always be with us in a world that is not—and never will be—Utopia; but if Isaiah could look with us into the future, he would see the great army of American workers enjoying their labors amid circumstances which are the prerequisites for a longer, healthier, happier life. To realize that obligation, industrial medicine is charged with the greater share of the responsibility. For the physician the call is clear, the opportunity abundant, and the field diversified.

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# CHAPTER II

# INDUSTRIAL MEDICINE AND WORKMEN'S COMPENSATION

Industrial medicine is not synonymous with "compensation medicine." This point has been thoroughly established in the preceding chapter, but it still bears repeating, since a large part of our profession is not aware of the distinction.

But medicine, whether it be industrial or general, has an inescapable responsibility in the equitable administration of workmen's compensation. Workmen's compensation, if properly dispensed, is one of the noblest forms of legislation ever enacted in this country. Unfortunately, the ultimate purpose of the act is not always attained and its intended benefits have not been extended uniformly to all workmen throughout the nation. Close examination of the laws of the various states will reveal a wide divergence in the benefits allowed.

Nor is the attitude of the medical profession toward the claimant for compensation uniform in its impartiality. A fairly large group of physicians are unacquainted with the intention of the act, and this ignorance often leads to injustice in the eventual determination of claims. Some physicians have developed a prejudice to the effect that the majority of compensation claims are unfounded, and almost automatically deny their probability, while an equally large group tends to classify all claims as occupational without endeavoring to determine their actual cause.

It is impossible to appreciate fully the purpose of workmen's compensation unless its historical background is clearly understood. With the inception of modern industry, the intimate relationship between master and servant disappeared. The master was no longer accessible to the servant, and his responsibility to the servant was allocated to so many underlings that the interests of the individual workman became lost in a numerical maze. Furthermore, modern industry increased the hazards incident to work; death, disabling injuries, or prolonged illness increased with the growth and diversity of industry. Yet, liability for these industrial injuries was not adequately assumed by the employer. Feeble attempts were made to rectify this irresponsible situation, but no decisive strides were made until 1906, when the Employer's Liability Act was passed. However, this act failed to benefit the ones for whom it was designed, since the injured workman had to prove negligence on the part of the employer, invariably a most difficult task to accomplish in a common court and against a shrewd company attorney. It was soon realized that the Employer's Liability Act insured the wrong party; namely, the employer.

By 1910 the mounting pressure from various socially minded agencies brought about passage of the first Workmen's Compensation Act, the major improvement of which was the establishment of the principle that "liability is without fault." At this writing, all states except Mississippi have some form of workmen's compensation. To the logical mind it is not apparent why one state will compensate for an illness due to benzol intoxication while a neighboring state will not. But as a matter of fact there is a wide divergence in the coverage of the act. Only sixteen states and the District of Columbia have established general coverage for occupational diseases; sixteen other states provide a "schedule coverage." The states with general coverage or an all-inclusive system are California, Connecticut, Florida, Illinois, Indiana, Massachusetts, Michigan, Minnesota, Missouri, Nebraska, New York, North Dakota, Ohio, Oregon, Washington, Wisconsin. Those having a schedule provision are Arizona, Arkansas, Colorado, Delaware, Idaho, Kentucky, Maine, Maryland, New Jersey, New Mexico, North Carolina, Pennsylvania, Rhode Island, Utah, Virginia, West Virginia.

In addition to the divergencies in the coverage system, there are also a number of other essential differences, only a few of which, however, are pertinent to this chapter:

1. At the moment, no compensation act undertakes to cover all types of employment.[1] The usual exemptions are (1) the small employer; (2) agriculture; (3) the domestic servant; (4) the casual laborer; (5) persons not gainfully employed; and (6) public employees. Regarding the numerical exception, some states such as California make no exception, requiring that compensation be compulsory for all employees, whether one or more. Other states begin compensation at various numbers such as three or more employees, five or more employees, etc. Almost every state excludes farm labor, although farming with its modern machinery is one of the most hazardous of all employments. Likewise, domestic help is excluded from coverage in most states. In Indiana, blind workers are not covered; in Iowa, clerical workers remain unprotected. In Maryland, the exception reads: "Farm labor, domestic servants and country blacksmiths, wheelrights, or similar rural employments, and employees not engaged in extra hazardous employment." Just how the geographic line should be drawn between a rural and an urban blacksmith only a Maryland court can be deemed competent to establish. Nor does there seem to be any equity in the distinction between hazardous and extra hazardous employments.

2. The Schedule System. In states where the schedule system is in effect, only specific occupations are covered. Those who defend this system maintain that its value over the all-inclusive system lies in the specific definitions it contains, as well as in its avoidance of unnecessary or prolonged litigation. But this contention fails to do justice to the possibility of exposure of a workman not engaged in the sched-

uled employment. For instance, a grinder may be covered by the act, while a man who is employed across the aisle or at the next bench in a different occupation is not covered, although he is exposed to practically the same environment as the grinder. And the schedule system does not recognize the possible inadequacy of hygienic or engineering measures.

The proponents of the schedule system contend that if a certain occupation in the course of time proves to be productive of a definite type of occupational disease, this disease can be included in the act at any subsequent legislative session. However, there is no assurance that such an enactment will always take place. Moreover, if it is the purpose of the act to provide medical care and compensation for all workmen, without exclusion of any one disease, it should not be necessary that one or more workmen become martyrs before the law is changed to benefit those subsequently exposed.[2]

3. The All-Inclusive System. In the states where this system has been enacted, no exceptions are made to any diseases or injuries as long as such "arise out of or in the course of employment." This is truly the ideal coverage, and yet it is not free from certain drawbacks. The primary objection lies in the very features which the framers of the act wished to avoid; namely, prolonged and expensive litigation. The all-inclusive system permits the appellate procedure. In Bulletin No. 672,[3] published by the United States Department of Labor in 1940, page 128, we read these words: "Those who reap the profits of litigation have in many localities led workers to overestimate the value of their constitutional right to court controversy in compensation cases, and the worker has seldom examined the argument either as to its applicability to compensation procedure or to the costs of such 'rights' to him." Thus the all-inclusive system not only permits the workman to become a vicitim of the unscrupulous attorney but also leaves him unprotected against the unwarranted vacillation on the part of the insurance carrier or physician.

✓ There are other variations in the act as it is administered in the several states, such as the exclusive or competitive State Fund, the Second Injury Fund, the System of Computing Compensation, etc. None of these sufficiently involve the physician to need further discussion.

One phase of the act, however, is of such controversial nature that it warrants a short comment. Labor unions and a section of the medical profession object to that portion of the act which forbids the workman the free choice of his physician. At the present time, one of the following variations always exists in the provision for medical care: (1) choice of the physician by the employer or insurance carrier; (2) selection by the employee from a list or panel compiled by the employer or insurance carrier; (3) selection by the worker from a panel made by the Industrial Accident Commission and medical societies; and (4) free choice by the worker. In some jurisdictions where the employer is legally entitled to select the physician, it is not uncommon for him to permit the worker the free choice of his own physician. Where this privilege is voluntarily accorded, it has been the experience that the workers frequently select the employer's medical service, since they have found this type of care satisfactory on previous occasions.

While organized medicine has always insisted on the "free choice of physician," it must be pointed out that under the exigencies created by the workmen's compensation act this is by no means always to the advantage of the workman. Many experienced physicians as well as laymen are opposed to the free choice of physician in workmen's compensation, and they advance the following reasons:

1. Only in rare cases is the workman able to distinguish good from bad medicine. If he were, or *if the majority of all people were so endowed,* the quack and irregular practitioner would go out of business overnight. It is even conceivable that many physicians who now refuse to take refresher courses in medicine would soon overcrowd postgraduate programs if the average man could tell the learned and capable doctor from his less-qualified counterpart.

2. The sweeping demand that workmen be permitted to choose any doctor also takes it for granted that industrial medicine requires no special training or experience. This assumption, however, is a grave error. There can be no justification for allowing an unwitting workman to drift to the incompetent physician, whether he be in or out of the regular practice.

3. It is not consistent that a large group of physicians who refuse to acquaint themselves with the special problems of industrial medicine still demand the right to diagnose or treat occupational diseases. When the practicing physician educates himself in this field, when our medical schools make a point of teaching industrial medicine and include it adequately in their postgraduate courses, then only will the objection to the present system be justified.

Incompetence in medical practice cannot be eliminated, but it can certainly be avoided to a greater extent under the present terms of workmen's compensation than in general medical practice. No necessity exists for discrimination against any physician who can demonstrate a knowledge of industrial medicine, who practices sound medical science, and who wishes to apply his knowledge within the framework of workmen's compensation.

To determine whether an alleged industrial injury or disease is of occupational origin requires an interpretation of the basic phrase of the act. Liability under workmen's compensation is created by the standardized phrase taken bodily from the English act and incorporated in the majority of the state laws of this country. Condensed, this phrase reads: "A case is compensable when it is a personal injury by accident arising out of and in the course of employment." Some states construe the word "accident" literally to convey only the meaning of trauma, whereas other states have broadened this meaning to include injury to health by an occupational disease. In the following discussion the entire phrase will be analyzed by the separate consideration of each one of its segments.

**Personal Injury.**—"Personal injury" means physical harm only. The purpose of compensation is not to reimburse one for a financial loss or wrongs committed against the reputation, social position, or other sensibilities of the employee. The term has been enlarged in scope in many states to mean that it is not necessary that a blow, fall, or actual violence occur to classify the case as compensable. Through the door of this revised interpretation have entered the occupational diseases in some states. Injury to the body can be sustained from noxious gases, dusts, metals, and the like. Many states having no schedule for occupational diseases have, on occasion, accepted a case as compensable upon this basis.

**By Accident.**—Here again it is an attempt to differentiate between a diseased condition and trauma resulting from work. This term places emphasis upon the time interval between the alleged injury and the onset of disability. Obviously a laceration or fracture or severe contusion is apparent to all, and medical aid is immediately sought. In contrast exists the *occupational illness*, which usually develops insidiously and which is not immediately apparent. The effect of an accident may be *far reaching* and the ultimate result foreign to the expected one. For example, compensation was granted by the Supreme Court of Pennsylvania in a case in which a workman was thrown violently against an object, fracturing a rib. Pleurisy developed, and in the course of this complication the patient became delirious and committed suicide. It was held that each subsequent event was the result of the original injury. The eventual results from an accident may also be influenced by *unusual factors* requiring an opinion as to whether the end result is compensable. In Wisconsin a claimant in the course of his employment cut his hand. He was given medical attention, and the wound was practically healed when he engaged in a boxing match. As a result his injury became infected and ended in the loss of the hand. The Supreme Court sustained the decision of the Industrial Commission that the accident was not the cause of the ultimate disability.

**Arising Out of and in the Course of Employment.**—This refers to the *time, place,* and *circumstance* under which the accident, or disease, takes place. The first portion of this phrase connotes or implies the danger peculiar to the work, and under this portion of the phrase must be considered most of the occupational diseases. It rules out conditions common to the neighborhood or district, or conditions which may occur at home as well as at work. Employees spend the greater part of each day at their work, and illness of any type may have its onset while they are at their employment. But the fact that it arises while one is at work does not mean that it arises *out* of the work. This fact is too often lost sight of in regard to backache or hernia, especially. This term "arising out of" implies that it must be incidental to the work and not independent of the relation of master to servant. It need not have been foreseen or expected, but after the event it must appear to have had its origin in the employment. Such an understanding enables one to render a just opinion regarding sunstroke or heat exhaustion or frostbite.

**In the Course of Employment.**—An injury can occur in the course of employment only while the workman is engaged in rendering service to his employer under agreement of hire. If, after his day's work is done, an employee of his own volition remains to visit others on another shift and suffers an injury, it is not compensable. Should a truck driver go out of his regular route for reasons personal to himself and not in the pursuit of his work, he would not be compensated for any injury he sustained while deviating from the work he was hired to do.

In addition to this basic phase of the act, it contains numerous other portions which directly or indirectly involve the practice of medicine. For the interpretation of these various portions, the writer is indebted to Douglas Campbell,[4] and the following excerpts are taken from his masterpiece in two volumes. While it is true that these definitions have been formulated with the California Act in mind, nevertheless in general and with few exceptions they are also applicable to workmen's compensation acts in other states.

At the end of this chapter will be found a number of charts which contain information for reference but which need no general discussion.

"*Physical Condition of Employee At Time of Injury No Bar to Compensation.*—An injured employee is entitled to compensation for disability proximately caused by an industrial injury regardless of whether his condition at the time of such injury be supernormal, average or subnormal. The theory of compensation liability and insurance therefore is based upon the law of averages and the view that the employer accepts the employee subject to his condition of susceptibility, except in cases in which a pre-existing disease is aggravated by injury, in which event apportionment of liability is in order."

"*Acts of God.*—As a general principle, the employer is not responsible for injuries to his workmen by lightning, storms, sunstroke, freezing, earthquake, floods and similar causes. These are considered as '*force majeure*' which human vigilance and industry can neither foresee nor prevent. The victim alone must bear such burden, inasmuch as human industry has nothing to do with it and the employee is no more subject thereto than any other person. For an injury or death to be compensable because of the exposure of the employee to the danger of the elements, the character of the employment itself must be shown to be of such a nature that it intensifies the risk and subjects the employee to a hazard greater than that faced by others in the same locality.

"In order to escape liability for an injury on the basis that it was caused by an 'Act of God', the latter must be the sole cause of the injury, unassisted by human agency. An 'Act of God' is an act of Nature, which implies entire exclusion of human agency. If an event occurs in any way through the intervention of man, it cannot be said to be an act of God. The principle embodied in all of the definitions is that the act must be occasioned exclusively by the violence of Nature, and all human agency is excluded from creating or entering into the cause of the mischief. When the cause is found to be in part the result of the participation of man, whether from active intervention, neglect, or failure to act, the whole occurrence is thereby humanized and removed from the operation of the rules applicable to the acts of God.

"The employment must specially expose the employee to such danger before an injury from the following causes becomes compensable: lightning, earthquake, wind storm, sunstroke, heat prostration, freezing, bites of ticks, dog, spider, mosquito, rooster, or other animal or insect."

"*Heat Stroke.*—The question as to whether heat stroke is to be regarded as an accidental injury within the meaning of Compensation Acts has been frequently before the courts. In some cases the distinction is made between injuries caused by natural and those caused by artificial heat. In other cases, it is said that the injury must be caused by some unusual or extraordinary condition.

"Heat stroke or prostration is an injury to the physical structures of the body and is compensable if the necessary circumstances of industrial connection or special exposure is established. In general, where an employee is required to do strenuous work in the sun, he is exposed to the danger of being overcome by the climatic conditions existing at the time, and his exposure is greater than that of the rest of the community. Similarly, when the employee is working indoors but, due to the construction of the building or the nature of his duties, is specially exposed by his environment to prostration from either artificial or natural heat, the resulting disability or death is compensable.

"The rule supported by the weight of authority seems to be, however, that heat prostration which results from the employment, whether or not due to unusual or extraordinary conditions, is to be deemed an injury within the meaning of the Act. Injury due to the heat alone ordinarily is not compensable; but when caused by heat plus the nature of the employment and the conditions under which the work is performed, it may fairly be said to arise out of the employment."

"*Compensation—Includes Medical Treatment.*—The term 'compensation' as used in the Act includes medical treatment as well as indemnity. The employer or his insurance carrier must provide: 'such medical, surgical and hospital treatment, nursing, medicines, medical and surgical supplies, crutches and apparatus, including artifiicial members, as may reasonably be required to cure and relieve from the effects of the injury.' "

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**U. S. DEPARTMENT OF LABOR**
**Division of Labor Standards**

## WORKMEN'S COMPENSATION—FATAL INJURIES
### Statutory Benefits for Widows and Children—June 1, 1946

***Chart I***

| State | Maximum period[1] | Maximum per week | | Maximum amounts[2] | | Minimum per week | | Percentage of wage | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | Widow only | Widow plus children | Widow only | Widow plus children | Widow only | One child only | Widow only | One child only | Maximum |
| Alabama | 300 weeks | $18.00 | $18.00 | $6,000 | $6,000 | [4]$ 5.00 | [4]$ 5.00 | 35 | 35 | 65 |
| Alaska | Not specified | (3) | (3) | 4,500 | 9,000 | | | | | |
| Arizona | do | (3) | (3) | (3) | (3) | | | 35 | 25 | 66⅔ |
| Arkansas | 450 weeks | 20.00 | 20.00 | 7,000 | 7,000 | 7.00 | 7.00 | 35 | 50 | 65 |
| California | Not specified | 25.00 | 25.00 | 6,000 | 6,000 | 6.50 | 6.50 | 61¾ | 61¾ | 61¾ |
| Colorado | 312 weeks | 14.00 | 14.00 | 4,375 | 4,375 | 5.00 | 5.00 | 50 | 50 | 50 |
| Connecticut | do[5] | 30.00 | 30.00 | 9,360 | | 7.00 | 7.00 | 50 | 50 | 50 |
| Delaware | do | 10.00 | 22.75 | 3,120 | | 10.00 | 2.50 | 25 | 25 | 65 |
| District of Columbia | Not specified | 13.13 | 25.00 | 7,500 | 7,500 | 4.20 | 1.80 | 35 | 15 | 66⅔ |
| Florida | 350 weeks | 22.00 | 22.00 | 5,000 | 5,000 | [4] 8.00 | [4] 8.00 | 35 | 25 | 60 |
| Georgia | 300 weeks | 17.00 | 17.00 | 5,100 | 5,100 | | | (6) | (6) | (6) |
| Hawaii | Widowhood | 15.00 | 25.00 | 7,500 | 7,500 | 4.80 | 3.60 | 40 | 30 | 66⅔ |
| Idaho | 400 weeks [5] | 12.00 | 12.00 | 4,800 | | [4] 8.00 | [4] 6.00 | 45 | 25 | 55 |
| Illinois | Not specified | 18.00 | 24.00 | 4,800 | 6,600 | 13.20 | 13.20 | 50 | 55 | 65 |
| Indiana | 350 weeks | 20.08 | 20.08 | 7,500 | 7,500 | [4]11.00 | [4]11.00 | 55 | 55 | 55 |
| Iowa | 300 weeks | 18.00 | 18.00 | 5,400 | 5,400 | [4] 8.00 | [4] 8.00 | 60 | 60 | 60 |
| Kansas | Not specified | 18.00 | 18.00 | 4,000 | 4,000 | | | | | |
| Kentucky | 400 weeks | 15.00 | 15.00 | 6,000 | 6,000 | 5.00 | 5.00 | 65 | 65 | 65 |
| Louisiana | 300 weeks | 20.00 | 20.00 | 6,000 | 6,000 | [4] 3.00 | [4] 3.00 | 32½ | 32½ | 65 |
| Maine | do | 21.00 | 21.00 | 5,000 | 5,000 | 7.00 | 7.00 | 66⅔ | 66⅔ | 66⅔ |
| Maryland | 416 weeks | 20.00 | 20.00 | 6,500 | 6,500 | [4]10.00 | [4]10.00 | 66⅔ | 66⅔ | 66⅔ |
| Massachusetts | 400 weeks[5] | 15.00 | (6) | 6,000 | | 15.00 | 15.00 | | | |
| Michigan | do | 19.00 | 27.00 | 7,600 | 10,800 | 8.00 | 8.00 | 66⅔ | 66⅔ | 66⅔ |
| Minnesota | Not specified | 24.00 | 24.00 | 7,500 | [7]7,500 | [4]10.00 | [4]10.00 | 40 | 45 | 66⅔ |
| Missouri | do | 20.00 | 20.00 | (3) | (3) | 6.00 | 6.00 | 66⅔ | 66⅔ | 66⅔ |
| Montana | 400 weeks | 15.00 | 21.00 | 6,000 | 8,400 | 8.00 | 8.00 | 50 | 50 | 66⅔ |
| Nebraska | 325 weeks | 18.00 | 18.00 | 5,850 | 5,850 | [4] 6.00 | [4] 6.00 | 66⅔ | 66⅔ | 66⅔ |
| Nevada | Not specified | 9.23 | 19.38 | (3) | (3) | | | 33⅓ | 30 | 70 |
| New Hampshire | do | (3) | (3) | 5,400 | 5,400 | | | | | |
| New Jersey | 300 weeks [5] | 25.00 | 25.00 | 7,500 | | 10.00 | 10.00 | 35 | 35 | 60 |
| New Mexico | do | 16.00 | 18.00 | 4,800 | 5,400 | 10.00 | 10.00 | 40 | 25 | 60 |
| New York | Not specified | 12.60 | 28.00 | (3) | (3) | 5.40 | 2.70 | 30 | 15 | 66⅔ |
| North Carolina | 350 weeks | 21.00 | 21.00 | 6,000 | 6,000 | 7.00 | 7.00 | 60 | 60 | 60 |
| North Dakota | Not specified | 12.25 | 23.33 | (3) | (3) | 8.75 | 6.25 | 35 | 25 | 66⅔ |

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| Ohio | 416 weeks | 24.50 | 24.50 | 7,500 | 7,500 | ---------- | ---------- | 66⅔ | 66⅔ | 66⅔ |
| Oregon | Not specified | 9.23 | 20.31 | (3) | (3) | 9.23 | 4.62 | ---------- | ---------- | ---------- |
| Pennsylvania | 300 weeks [5] | 12.00 | 20.00 | 3,600 | ---------- | 7.48 | 4.25 | 44 | 25 | 66⅔ |
| Puerto Rico | Not specified | (3) | (3) | 3,500 | 3,500 | ---------- | ---------- | ---------- | ---------- | ---------- |
| Rhode Island | 600 weeks | 16.00 | 20.00 | 9,600 | 12,000 | 12.00 | 12.00 | 60 | 60 | 60 |
| South Carolina | 350 weeks | 25.00 | 25.00 | 6,000 | 6,000 | 5.00 | 5.00 | 60 | 60 | 60 |
| South Dakota | Not specified | (3) | (3) | 5,000 | 5,000 | ---------- | ---------- | ---------- | ---------- | ---------- |
| Tennessee | 400 weeks | 18.00 | 18.00 | 5,000 | 5,000 | [7] 7.00 | [7] 7.00 | 35 | 30 | 60 |
| Texas | 360 weeks | 20.00 | 20.00 | 7,200 | 7,200 | 7.00 | 7.00 | 60 | 60 | 60 |
| Utah | 313 weeks | 22.50 | 22.50 | 7,000 | 7,000 | ---------- | ---------- | 60 | 60 | 60 |
| Vermont | 260 weeks | 13.46 | 13.46 | 3,500 | 3,500 | 5.60 | 6.30 | 40 | 45 | 50 |
| Virginia | 300 weeks | 20.00 | 20.00 | 6,600 | 6,600 | 6.00 | 6.00 | 60 | 60 | 60 |
| Washington | Not specified | 11.54 | (8) | (3) | (3) | 11.54 | 5.77 | ---------- | ---------- | ---------- |
| West Virginia | do | 6.92 | (9) | (3) | (3) | 6.92 | 3.46 | ---------- | ---------- | ---------- |
| Wisconsin | 400 weeks [10] | 18.50 | 18.50 | 7,400 | (10) | 10.00 | 10.00 | 50 | 50 | 50 |
| Wyoming | Not specified | 12.69 | (11) | 4,950 | 11,000 | 12.69 | 3.81 | ---------- | ---------- | ---------- |
| United States: | | | | | | | | | | |
| Longshoremen | do | 13.13 | 25.00 | 7,500 | 7,500 | 4.20 | 1.80 | 35 | 15 | 66⅔ |
| Civil employees | do | 14.13 | 26.92 | ---------- | ---------- | 7.07 | 5.04 | 35 | 25 | 66⅔ |

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[1] Benefits are generally payable to widow until remarriage and to children until specified age, but not in excess of maximum period or amount stated in law. A few laws also provide lump sums payable to widow upon remarriage. In some States benefits continue for longer period in case of incapacitated children.

[2] Total maximum payments computed where not stipulated by law. Disability payments deducted in all laws except those of Alaska, Arizona, Arkansas, Delaware, District of Columbia, Hawaii, Michigan, Missouri, Nevada, New York, North Dakota, Oregon, Washington, West Virginia, Wisconsin, Wyoming, and United States (Longshoremen).

[3] No limit.

[4] Actual wage, if less.

[5] Thereafter to children until specified age.

[6] 85% of compensation provided for total disability.

[7] Thereafter compensation from special fund, maximum $15, until youngest child is 18 or total additional payments amount to $2,500.

[8] $11.54 for widow, plus $3.46 for first child, $2.31 for second child, and $1.73 for each additional child.

[9] $6.92 for widow plus $2.31 for each child.

[10] 780 weeks for children. Children's benefits from State fund plus widow's benefits may exceed $20,000.

[11] $12.69 for widow, plus $3.81 for each child. Maximum for children $6,050.

**U. S. DEPARTMENT OF LABOR**
**Division of Labor Standards**

## WORKMEN'S COMPENSATION—BENEFITS FOR PERMANENT AND TEMPORARY TOTAL DISABILITIES—JUNE 1, 1946

***Chart II***

| State | Limitations on permanent total | | | | | Limitations on temporary total | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | Maximum percentage of wages | Maximum weekly payment | Minimum weekly payment | Time limit | Amount limit[1] | Maximum percentage of wages | Maximum weekly payment | Minimum weekly payment | Time limit | Amount limit[1] |
| Alabama | 65 | $18; $5 (actual wage if less) after 400 weeks. | $5 (actual wage if less). | 550 weeks | $6,000 | 65 | $18 | $5 (actual wage if less) | 300 weeks | $5,400 |
| Alaska | | | | | 9,000 | 65 | | | Period of disability | |
| Arizona | [2]65 | | | Life | | 65 | | | 433 weeks | |
| Arkansas | 65 | $20 | $7 | 450 weeks | 7,000 | 65 | $20 | $7 | 450 weeks | 7,000 |
| California | 61¾ | $30 | $6.50 | Life; 40 percent of wages after 240 weeks. | | 61¾ | $30 | $6.50 | 240 weeks | 6,000 |
| Colorado | 50 | $14 | $5 | Life | | 50 | $14 | $5 | Period of disability | |
| Connecticut | 50 | $30 | $7 | 520 weeks | 15,600 | 50 | $30 | $7 | 520 weeks | 15,600 |
| Delaware | 60 | $21 | $8 (actual wage if less). | 500 weeks | 10,500 | 60 | $21 | $8 (actual wage if less). | 500 weeks | 10,500 |
| Dist. of Columbia | 66⅔ | $25 | do | Period of disability | 7,500 | 66⅔ | $25 | do | Period of disability | 7,500 |
| Florida | 60 | $22 | do | 350 weeks | 5,000 | 60 | $22 | do | 350 weeks | 5,000 |
| Georgia | 50 | $20 | $4 (actual wage if less). | do | 7,000 | 50 | $20 | $4 (actual wage if less). | do | 7,000 |
| Hawaii | 66⅔ | $25[2] | $8 less). | Period of disability | 7,500 | 66⅔ | $25 | $8(actual wage if less). | Period of disability | 7,500 |
| Idaho | 60 | $14; $16 for married employee, plus $1 for each child, maximum $20. | $8; $10 for employee with children. | Life; $8 per week after 400 weeks. | | 60 | $14; $16 for married employee, plus $1 for each child, maximum $20. | $8; $10 for employee with children. | 400 weeks; thereafter $8 per week. | |
| Illinois | 65 | $18; $19.20 if 2 children; $21.60 if 3; and $24 if 4 or more children. | $9.00 | Life | [3]6,600 | 65 | $18; $19.20 if 2 children; $21.60 if 3; and $24 if 4 or more children. | $9.00 | Period of disability | 6,600 |
| Indiana | 55 | $20.08 | $11 (actual wage if less). | 500 weeks | 7,500 | 55 | $20.08 | $11 (actual wage if less). | 500 weeks | 7,500 |

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| Iowa | 60 | $18 | $8 (actual wage if less). | 400 weeks | 7,200 | 60 | $18 | $8 (actual wage if less). | 300 weeks | 5,400 |
| Kansas | 60 | $18 | $6 | 416 weeks | 7,488 | 60 | $18 | $6 | 416 weeks | 7,488 |
| Kentucky | 65 | $18 | $5 | 520 weeks | 9,000 | 65 | $18 | $5 | 520 weeks | 9,000 |
| Louisiana | 65 | $20 | $3 (actual wage if less). | 400 weeks | 8,000 | 65 | $20 | $3 (actual wage if less). | 300 weeks | 6,000 |
| Maine | 66⅔ | $21 | $7 | 500 weeks | 7,500 | 66⅔ | $21 | $7 | 500 weeks | 7,500 |
| Maryland | 66⅔ | $23 | $10 (actual wage if less). | Period of disability | 7,500 | 66⅔ | $23 | $10 (actual wage if less). | 312 weeks | 3,750 |
| Massachusetts | 66⅔ | $25; plus $2.50 for each dependent; total benefits limited to weekly wage. | $18 (actual wage if less). | Life | [4]10,000 | 66⅔ | $25; plus $2.50 for each dependent; total benefits limited to weekly wage. | $15 (actual wage if less). | Period of disability | 10,000 |
| Michigan | 66⅔ | $21 | $10 | 750 weeks | 15,750 | 66⅔ | $21 | $10 | 500 weeks | 10,500 |
| Minnesota | 66⅔ | $24 | $10 (actual wage if less). | Period of disability | [5]10,000 | 66⅔ | $24 | $10 (actual wage if less). | 300 weeks | 7,200 |
| Missouri | 66⅔ | $20 | $6 | 300 weeks; thereafter 25 percent of wages for life. | | 66⅔ | $20 | $6 (actual wage if less). | 400 weeks | 8,000 |
| Montana | 66⅔ | $15; $17 if one dependent; $18 if 2; $19 if 3; $20 if 4; $21 if 5 or more dependents. | $8 | 500 weeks | 10,500 | 66⅔ | $15; $17 if one dependent; $18 if 2; $19 if 3; $20 if 4; $21 if 5 or more dependents. | $8 | 300 weeks | 6,300 |
| Nebraska | 66⅔ | $18 | $6 (actual wage if less). | 300 weeks; thereafter 45 percent of wages for life; maximum $12, minimum $4.50 (actual wage if less). | | 66⅔ | $18 | $6 (actual wage if less). | do | 5,400 |
| Nevada | 60 | $13.85[2] | $6.92 | Life | | 66⅔ | $18.46, plus $2.31 for total dependents residing in the United States. | $6.92 | 433 weeks | 8,000 |
| New Hampshire | 50 | $21 | $8 | 300 weeks | 6,400 | 50 | $21 | $8 | 300 weeks | 6,300 |
| New Jersey | 66⅔ | $25 | $10 | 450 weeks; thereafter at reduced rate during rehabilitation. | | 66⅔ | $25 | $10 | do | 7,500 |
| New Mexico | 60 | $18 | $10 (actual wage if less). | 550 weeks | 9,900 | 60 | $18 | $10 (actual wage if less). | 550 weeks | 9,900 |
| New York | 66⅔ | $28[6] | $15 (actual wage if less). | Life | | 66⅔ | $28[6] | $12 (actual wage if less). | Period of disability | 5,000 |
| North Carolina | 60 | $21 | $7 | 400 weeks | 6,000 | 60 | $21 | $7 | 400 weeks | 6,000 |
| North Dakota | 66⅔ | $20 | $9 | Life | | 66⅔ | $20, plus $2 for each child under 18, maximum $30. | $9 | Period of disability | |

See footnotes at end of chart.

**U. S. DEPARTMENT OF LABOR**
**Division of Labor Standards**

## WORKMEN'S COMPENSATION—BENEFITS FOR PERMANENT AND TEMPORARY TOTAL DISABILITIES—JUNE 1, 1946

***Chart II—Cont'd***

| State | Limitations on permanent total | | | | | Limitations on temporary total | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | Maximum percentage of wages | Maximum weekly payment | Minimum weekly payment | Time limit | Amount limit[1] | Maximum percentage of wages | Maximum weekly payment | Minimum weekly payment | Time limit | Amount limit[1] |
| Ohio | 66⅔ | $24.50[7] | $10 (actual wage if less). | do | | 66⅔ | $24.50[7] | $10 (actual wage if less). | 312 weeks | $ 4,200 |
| Oklahoma | 66⅔ | $21 | do | 500 weeks | $10,500 | 66⅔ | $21 | do | 300 weeks | 6,300 |
| Oregon | | $9.23; $11.54 for married employee plus $2.77 for first 2 children and $1.85 for additional children, maximum $20.31. | | Life | | 66⅔ | $15; $17.31 if married; $19.61 if one child; $21.92 if 2 children; $24.23 if 3 children; $26.54 if 4 or more children. | $6.92 (actual wage if less). | Period of disability | |
| Pennsylvania | 66⅔ | $20 | $10 (actual wage if not under $5). | 500 weeks | 10,000 | 66⅔ | $20 | $10 (actual wage if not under $5). | 500 weeks | 10,000 |
| Puerto Rico | 50 | $15 | $4 | 340 weeks | 3,000 | 50 | $15 | $3 | 104 weeks | 1,560 |
| Rhode Island | 60 | $20 | $12 | 1,000 weeks | 12,000 | 60 | $20 | $12 | 1,000 weeks | 12,000 |
| South Carolina | 60 | $25 | $5 | 500 weeks | 6,000 | 60 | $25 | $5 | 500 weeks | 6,000 |
| South Dakota | 55 | $15 | $7.50 | Period of disability | 5,000 | 55 | $15 | $7.50 (actual wage if less). | 312 weeks | 4,680 |
| Tennessee | 60 | $18 | $7 (actual wage if less). | 400 weeks; thereafter $7 (actual wage if less) for additional 150 weeks. | 5,000 | 60 | $18 | $7 (actual wage if less). | 300 weeks | 5,400 |
| Texas | 60 | $20 | $7 | 401 weeks | 8,020 | 60 | $20 | $7 | 401 weeks | 8,020 |
| Utah | 60 | $22.50 plus 5 percent for each child (not to exceed 5). | $10 | 260 weeks; thereafter 45 percent of wages during disability. | 8,500 | 60 | $22.50 plus 5 percent for each child (not to exceed 5). | $10 (actual wage if less). | 313 weeks | 8,500 |
| Vermont | 50 | $20 | $10 (actual wage if less). | 260 weeks | 5,200 | 50 | $20 | $10 (actual wage if less). | 260 weeks | 5,200 |
| Virginia | 60 | $20 | $6 | 500 weeks | 7,800 | 60 | $20 | $6 | 500 weeks | 7,800 |
| Washington | | $11.54; $13.85 for married employee plus $3.46 for youngest child; $2.31 for next child; $1.73 for each additional child.[2] | | Life | | | $11.54; $13.85 for married employee plus $3.46 for youngest child; $2.31 for next child; $1.73 for each additional child.[8] | | Period of disability | |

|  |  |  |  |  |  |  |  |  |  |  |
|---|---|---|---|---|---|---|---|---|---|---|
| **West Virginia** | 66⅔ | $18 | $10 | do | | 66⅔ | $18 | $10 | 156 weeks | 2,808 |
| **Wisconsin** | 70 | $25.90 | $14 | do | | 70 | $25.90 | $7.85 | Period of disability | |
| **Wyoming** | | $12.69; $17.77 for married employee, plus $3.81 for each child. | | Period of disability | [9]12,650 | | $12.69; $15.23 for married employee, plus $2.54 for each child; total maximum $27.92. | | do | |
| United States: | | | | | | | | | | |
| Civil employees | 66⅔ | $26.92[2] | $13.46 | Life | | 66⅔ | $26.92[2] | $13.46 | do | |
| Longshoremen | 66⅔ | $25 | $8 (actual wage if less). | Period of disability | 7,500 | 66⅔ | $25 | $8 (actual wage if less). | do | 7,500 |

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[1] Total maximum payments computed where not stipulated by law.

[2] Additional compensation for constant attendant if necessary.

[3] Thereafter annual pension for life of 12 percent of previous payments in specific case of permanent total disability and 8 percent of such amount in other cases.

[4] Thereafter 50 percent of wage, maximum $22, plus $2.50 for each dependent with total benefits limited to weekly wage.

[5] Thereafter 50 percent of wages payable out of special fund, maximum $15 and minimum $8 (actual wage if less). Maximum amount $2,500.

[6] When accident or disablement due to occupational disease occurred prior to June 1, 1944, the $28 maximum rate applies only to periods of total disability up to May 31, 1947.

[7] For period ending Sept. 30, 1947.

[8] Special schedule of monthly payments for first 6 months of disability for married or widowed employee ranging from $22.50 for childless woman whose husband is not an invalid to $75 for married workman or widow with two children plus $7.50 for each additional child. Additional allowance for constant attendant if necessary.

[9] Maximum for workman $6,600 and for children $6,050.

**U. S. DEPARTMENT OF LABOR**
**Division of Labor Standards**

# WORKMEN'S COMPENSATION—OCCUPATIONAL DISEASES

## Summary of Statutory Provisions—June 1, 1946

*(Because of Numerous Special Features Silicosis Provisions Are Covered Separately on Chart IV)*

***Chart III***

| State | Administrative agency | Application of law and coverage | Medical boards | Time limit on claim filing | Time limitation on eligibility for death benefits | Deductions from death awards | Medical care | Compensation |
|---|---|---|---|---|---|---|---|---|
| Arizona | Industrial Commission. | Compulsory for 35 listed diseases; employees may reject. | Board of 3 medical consultants may be appointed by commission. Report prima facie evidence of facts. | 90 days for benzol poisoning, 60 days for other diseases, and 6 months after death. | 1 year after last day of work or 3 years if following continuous disability. | Disability payments. | Maximum $500 | Total disability, two-thirds of wages, maximum $25, minimum $8. Death $12.50 weekly, plus $2.50 for dependents, maximum $25; total maximum $5,000. No compensation for partial disability. |
| Arkansas | Workmen's Compensation Commission. | Compulsory for 15 diseases or groups of diseases. | Board of 3 appointed by commission; findings conclusive. | 1 year after injury, death, last payment or lost damage suit. | 1 year after last exposure, or 7 years if following continuous disability. | No provision | 60-day limit; may be extended indefinitely. | Same as for accidents including partial disability. |
| California | Industrial Accident Commission in Department of Industrial Relations. | Compulsory for all diseases. | No provision | Disability, 6 months to 2 years after injury; death, 1 year after death and from 2 years to 240 weeks after injury. | 1 year after injury or 240 weeks if following continuous disability. | Disability payments if death occurs more than 12 months from injury. | Unlimited | Do. |
| Colorado | Industrial Commission. | Elective for 22 listed diseases and poisonings. | Panel of 10; examinations and findings made by one or more members; advisory only. | 90 days for benzol poisoning, 60 days for other diseases, and 6 months after death. | 1 year after exposure, or 3 years if following continuous disability. | Disability payments | Maximum $500 | Same as for accidents, but subject to maximum in all cases of $4,375. No compensation for partial disability, and no compensation during first 30 days of disability unless disability lasts 60 days or longer. |
| Connecticut | Commissioner in each of 5 districts. | Elective for all diseases. | No provision | 1 year after first manifestation; 2 years if death results in 2 years; not more than 5 years after leaving employment. | | Disability payments if death occurs more than 2 years after first manifestation. | Unlimited | Same as for accidents including partial disability. |
| Delaware | Industrial Accident Board. | Compulsory for 16 listed diseases. | do | 1 year after last exposure, last payment or default. | | No provision | Limited to 30 days and $200, may be extended indefinitely. | Do. |
| Dist. of Columbia | U. S. Employees' Compensation Commission. | Compulsory for all diseases. | do | 1 year after injury, death, or last payment. | | do | Unlimited | Do. |
| Florida | Industrial Commission. | Elective for all diseases. | do | do | Following continuous disability and within 350 weeks after last exposure. | Disability payments. | Maximum $1,000, may be extended. | Do. |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| **Georgia** | State Board of Workmen's Compensation. | Elective for 4 diseases or groups of diseases. | Board of 5 appointed by Governor, findings conclusive. | 1 year after injury or death. | No provision | do | Limited to 10 weeks and $500; may be extended. | Same as for accidents except that there is no compensation for partial loss or loss of use of a member or vision of an eye. |
| **Hawaii** | Bureau of Workmen's Compensation. | Compulsory for all diseases. | No provision | 3 months after injury or death. | 1 year after injury | No provision | Unlimited | Same as for accidents including partial disability. |
| **Idaho** | Industrial Accident Board. | Compulsory for 11 diseases or groups of diseases. | Panel of 3 appointed by the board, findings conclusive. | 1 year after disablement, death, or last payment. | 1 year after last exposure or 2 years if following continuous disability. | Disability payments | 90 day limit, may be extended to 180 days. | Do. |
| **Illinois** | Industrial Commission. | Elective for all diseases. | No provision | 1 year after disablement or last payment, or if death occurs within said year, 1 year after death. | | Disability payments but with minimum compensation of $500. | Unlimited | Do. |
| **Indiana** | Industrial Board | do | do | 1 year after date of disablement or death. | 1 year after disablement. | Disability payments. | 30 day limit, may be extended to 60 days. | Do. |
| **Kentucky** | Workmen's Compensation Board. | Elective for a few specified diseases.[1] | Medical committee of 3 appointed by board with approval of Governor. | 1 year after injury, death, or last payment. | 2 years after injury | do | Limited to 90 days and $200, may be increased to $400. | Do. |
| Maine | Industrial Accident Commission. | Elective for 13 listed diseases. | No provision | 1 year after injury or death. | 1 year after exposure, or 7 years if following continuous disability. | Disability payments. | Maximum $100, may be extended. | $500 maximum first month act effective (January 1946), increased by $50 each month until maximum is same as for accidents. |
| Maryland | do | Compulsory for 39 listed diseases. | Board of 3 appointed by Governor; findings conclusive. | 1 year after disablement or death; delay excusable under certain circumstances. | do | Disability payments, but with minimum compensation of $1,000. | Limited to $750; $250 additional may be allowed for hospital services. | Same as for accidents including partial disability. |
| Massachusetts | Department of Industrial Accidents. | Compulsory for all diseases. | Department may appoint 3 medical referees; findings binding but subject to review by reviewing board.[2] | 6 months after injury or death; delay may be excused. | No provision | Disability payments. | Unlimited | Do. |
| Michigan | Department of Labor and Industry. | do | In disputed cases department must appoint 3 impartial physicians; report conclusive.[2] | 6 months after disablement or death. | do | | 6-month limit; may be extended for additional 6 months. | Do. |
| Minnesota | Industrial Commission. | do | Board of 3 appointed by commission; findings conclusive.[2] | 1 year after last exposure, last payment, or default. | do | Disability payments. | Unlimited | Same as for accidents, except for partial disability. |
| Missouri | Workmen's Compensation Commission. | Elective for all diseases. | | 1 year after injury, death, or last payment. | 300 weeks after injury. | do | Limited to 90 days and $750; may be extended. | Same as for accidents including partial disability. |

See footnotes at end of chart.

U. S. DEPARTMENT OF LABOR
Division of Labor Standards

## WORKMEN'S COMPENSATION—OCCUPATIONAL DISEASES
### Summary of Statutory Provisions—June 1, 1946
*(Because of Numerous Special Features Silicosis Provisions Are Covered Separately on Chart IV)*

*Chart III—Cont'd*

| State | Administrative agency | Application of law and coverage | Medical boards | Time limit on claim filing | Time limitation on eligibility for death benefits | Deductions from death awards | Medical care | Compensation |
|---|---|---|---|---|---|---|---|---|
| Nebraska | Workmen's Compensation Court. | do. | No provision | 6 months after injury or death; barred unless filed in 1 year. | 325 weeks after injury. | do. | Unlimited | Do. |
| New Jersey | Workmen's Compensation Bureau in Department of Labor. | Elective for 13 listed diseases. | do. | 1 year after last exposure, last payment, or default. | No provision | do. | Limited to $50 medical and $50 hospital; may be extended. | Do. |
| New Mexico | Courts | Elective for 30 listed diseases and poisonings.[3] | do. | 90 days for benzol poisoning, 60 days for other diseases, 6 months after death, and in all cases, 31 days after last payment. | 1 year after last exposure, or 3 years if following continuous disability. | do. | Maximum $400 | $15 a week, plus $1.50 per week for each dependent child in death cases; maximum $5,000. |
| New York | Chairman, Workmen's Compensation Board. | Compulsory for all diseases. | do. | 1 year after injury or death (Workmen's Compensation Board may extend to 2 years). | No provision | No provision | Unlimited | Same as for accidents including partial disability. |
| North Carolina | Industrial Commission. | Elective for 25 listed diseases. | Board of 3 appointed by commission; findings advisory. | 1 year after disablement, death, or last payment.[4] | 2 years after injury, or 6 years if totally disabled.[4] | do. | do. | Do. |
| North Dakota | Workmen's Compensation Bureau. | Compulsory for all diseases. | | 60 days after injury or death; barred after 1 year. | 6 years after injury | do. | do. | Do. |
| Ohio | Industrial Commission. | do. | Medical boards of review; findings conclusive if favorable to claimant. | 4 months after disability; 6 months after death. | No provision | Disability payments when total of disability payments and death award amounts to $7,500. | $200 limit may be increased by unanimous approval of commission. | Do. |
| Oregon | Industrial accident Commission. | Elective for all diseases. | Medical board of review to which claimant may appeal; decision final. | Disability, 3 months after injury; death, 1 year after injury. | No provision | No provision | Limited to $250; may be extended. | Do. |
| Pennsylvania | Workmen's Compensation Bureau in Department of Labor and industry. | Elective for 12 diseases or groups of diseases. | Board of 3 appointed by secretary of labor and industry; findings conclusive. | 1 year after disablement, death, or last payment. | 3 years from date of last employment. | Disability payments. | Medical, 60 days and $150; hospital, 60 days; may be extended 30 days, maximum $75. | Do. |
| Puerto Rico | State Insurance Fund. | Compulsory for 15 listed diseases and poisonings. | No provision | No provision | 2 years after injury | No provision | Unlimited | Do. |
| Rhode Island | Division of Workmen's Compensation in Department of Labor. | Elective for 31 listed diseases. | do. | 2 years after injury or death. | No provision | Disability payments. | $300 to $500 limit; may be extended. | Do. |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| **Utah** | Industrial Commission. | Compulsory for 27 listed diseases. | No provision | 60 days (90 for benzol) after cause of action arises; 6 months after death. | 1 year from last day actually worked; 3 years if continuous disability prior to death. | Disability payments. | Limited to $500 | Maximum $16 a week; in death cases additional allowance of 5 percent for each child up to $5; total $5,000. No compensation for partial disability. |
| **Virginia** | do | Elective for 46 listed diseases; full coverage optional. | do | 1 year after disability or death. | 6 years after injury | do | Limited to 60 days; may be extended 180 additional days. | Same as for accidents including partial disability. |
| **Washington** | Department of Labor and Industries. | Compulsory for all diseases. | do | (5) | No provision | No provision | Limited to compensable period; may be extended. | Do. |
| **West Virginia** | Workmen's compensation commissioner. | Silicosis only (see Chart VI for details). | | | | | | |
| **Wisconsin** | Industrial Commission. | Compulsory for all diseases. | No provision | 2 years from injury or death, or from date claimant knew or should have known nature of disability and relation to employment, with limit of 6 years from date of injury. | No provision | No provision | Unlimited | Same as for accidents, including partial disability. |
| **United States:** | | | | | | | | |
| **Civil Employees** | U. S. Employees' Compensation Commission. | do | do | Disability: 60 days after injury; may be extended to 1 year. Death: 1 year after death. | 1 year after injury, or 6 years if following continuous disability. | do | do | Do. |
| **Longshoremen** | do | do | do | 1 year after injury, death, or last payment. | No provision | do | do | Do. |

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[1] Covers only injury or death by gas or smoke in mines and poisonous gas in any occupation.

[2] This provision has been held unconstitutional.

[3] Applies to employers of 4 or more, except agricultural laborers and domestic servants.

[4] The limitations of claim filing and death for lead poisoning are the same as for silicosis. (See Chart IV.)

[5] By court decision limitation on claim filing is not applicable to occupational diseases.

**U. S. DEPARTMENT OF LABOR**
**Division of Labor Standards**

***Chart IV***

## WORKMEN'S COMPENSATION—SILICOSIS

### Summary of Statutory Provisions—June 1, 1946

| State | Administrative agency | Application of law | Medical boards | Time limit on claim filing | Time limitation on eligibility for death benefits | Deductions from death awards | Medical care | Partial disability | Special liability limitations | Requirements for exposure |
|---|---|---|---|---|---|---|---|---|---|---|
| Arizona | Industrial Commission. | Compulsory on part of employers; employees may reject. | 3 medical consultants appointed by commission; report is prima facie evidence of medical facts. | 1 year after disablement; 6 months after death. | 2 years after last exposure or 5 years if death follows continuous disablement. | Disability payments; award prorated for aggravation. | Maximum $500. | Not compensated. | $800 maximum first month act effective (March 1943); increased by $50 each month until maximum is $5,000. | 5 years' exposure in State in last 10 years. |
| Arkansas | Workmen's Compensation Commission. | Compulsory | Board of 3 appointed by commission. Medical findings conclusive. | 1 year after injury, death, last payment or lost damage suit. | 3 years after last exposure or 7 years if following continuous disablement. | do | 90-day limit; may be extended to 180 days. | Compensated if 33⅓ percent or more. | $500 maximum first month act effective (December 1940), increased by $50 each month until maximum is same as for accidents; 26 weeks' maximum benefits for nondisabled silicotics removed from employment. | 5 years' exposure in last 10; 2 of 5 in State.[2] |
| California[1] | Industrial Accident Commission in Department of Industrial Relations. | do | No provision | Disability, 6 months to 2 years after injury; death, 1 year after death and from 2 years to 240 weeks after injury. | 1 year after injury; or 240 weeks if following continuous disability. | Disability payments if death more than 12 months after injury; award prorated for aggravation. | Unlimited | Compensated | None | No provision. |
| Colorado | Industrial Commission. | Elective | Panel of 10, examinations and findings made by one or more members; advisory only. | 1 year after disablement; 6 months after death. | 2 years after last exposure or 5 years if following continuous disablement. | Disability payments; award prorated for aggravation. | Limited to $500. | Not compensated. | Maximum in all cases $4.375. No compensation during first 30 days of disability unless disability last 60 days or longer. | 5 years' exposure in State in last 10 years. |
| Connecticut[1] | Commissioner in each of 5 districts. | do | No provision | 1 year after first manifestation; 2 years, if death within 2 years; 5 years after leaving employment. | No provision | Disability payments if death more than 2 years after first manifestation; award prorated for aggravation. | do | Compensated | None | No provision. |
| Delaware[1] | Industrial Accident Board. | Compulsory | do | 1 year after last exposure, last payment, or default. | do | Prorated for aggravation. | Limited to 30 days and $200; may be extended. | do | do | Do. |

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|---|---|---|---|---|---|---|---|---|---|---|
| District of Columbia.[1] | U. S. Employees Compensation Commission. | do | do | 1 year after injury, death, or last payment. | do | No provision | Unlimited | do | do | Do. |
| Florida | Industrial Commission. | Elective | do | 1 year after injury, death, last payment, or lost damage suit. | 350 weeks after last exposure and following continuous disability. | Disability payments; award prorated for aggravation. | Limited to $1,000; may be extended. | Not compensated. | $500 maximum first month act effective (July 1945); increased by $50 each month until maximum is same as for accidents. | 5 years' exposure in last 10; 2 of 5 in State.[2] |
| Georgia | State Board of Workmen's Compensation. | do | Board of 5 appointed by Governor; findings conclusive. | 1 year after injury or death. | No provision | do | Limited to 10 weeks and $500; may be extended. | Compensated except for partial loss or loss of use of a member or vision of an eye. | $500 maximum first month act effective (May 1946); increased $50 each month until maximum is same as for accident. Partial waiver permitted for nondisabled silicotics. | Do. |
| Hawaii[1] | Bureau of Workmen's Compensation. | Compulsory | No provision | 3 months after injury or death; 5 years after accident or occurrence causing injury. | 1 year after injury. | No provision | Unlimited | Compensated | None | No provision. |
| Idaho | Industrial Accident Board. | Compulsory | Panel of 3 selected by Board from list of 6 appointed by Governor. | 1 year after disablement, death or last payment. | 2 years after last exposure. | Award prorated for aggravation. | 90-day limit; may be extended to 180 days. | Not compensated; discretionary payment in case of discharge or job termination due to nondisabling silicosis, not exceeding $750. | $500 maximum first month act effective (May 1939); increased by $50 each month until maximum is same as for accidents. Waivers permitted for nondisabled silicotics. | 5 years' exposure in last 10, last 2 of 5 in State.[2] |
| Illinois | Industrial Commission. | Elective | No provision | 1 year after disablement or last payment; or if death occurs within said year 1 year after death. | No provision | Disability payments but with minimum compensation of $500. | 6-month limit | Compensated | None | Disability within 3 years of last exposure. |
| Indiana | Industrial Board. | do | do | 1 year after date of disablement or death. | 1 year after disablement, or 300 weeks if following continuous disability. | Disability payments. | 30-day limit; may be extended to 60 days. | do | do | Do. |

See footnotes at end of chart.

## WORKMEN'S COMPENSATION—SILICOSIS

### Summary of Statutory Provisions—June 1, 1946

*Chart IV—Cont'd*

| State | Administrative agency | Application of law | Medical boards | Time limit on claim filing | Time limitation on eligibility for death benefits | Deductions from death awards | Medical care | Partial disability | Special liability limitations | Requirements for exposure |
|---|---|---|---|---|---|---|---|---|---|---|
| Kentucky | Workmen's Compensation Board. | Voluntary, by joint action. | Medical committee of 5 appointed by board with approval of Governor. | do | 3 years after last exposure or 7 years if following continuous disablement. | Disability payments, award prorated for aggravation. | 90 days and $200; may be increased to $400. | do | 26 weeks maximum benefits for nondisabled silicotics removed from employment. | 2 years' exposure in State in last 5 years. |
| Maryland | Industrial Accident Commission. | Compulsory | Board of 3 appointed by Governor, medical findings conclusive. | 1 year after disablement or death, delay excusable under certain circumstances. | do | Disability payments, but with minimum of $1,000, award prorated for aggravation. | Limited to $750; $250 additional may be allowed for hospital services. | Not compensated. | $500 maximum first month act effective (June 1939; ) increased by $50 each month until maximum is same as for accidents; in death cases period is life expectancy of deceased. Partial waiver for nondisabled silicotics. | 5 years' exposure in last 10; 2 of 5 in State.[3] |
| Massachusetts[2] | Department of Industrial Accidents. | Compulsory; special provisions for granite industry. | Department may appoint 3 medical referees; medical findings binding but subject to review by reviewing board.[4] | 6 months after injury or death, delay excusable. | 3 years after last exposure or 7 years, if following continuous disability beginning within 3 years. | Disability payments. | Unlimited | do | $500 maximum first month act effective (August 1939); or prior insurance policy effective; increased by $50 each month until maximum is $3,000. | 5 years' exposure in last 10; 1 of 5 in State; 180 days minimum exposure. |
| Michigan | Department of Labor and Industry. | Compulsory | In disputed cases department must appoint 3 impartial physicians; medical findings conclusive.[4] | 6 months after disablement or death. | No provision | Disability payments; award prorated for aggravation. | 6-month limit; may be extended for an additional 6 months. | do | $500 maximum first month act effective (October 1937); increased by $75 each month until maximum is $6,000.[5] | No provision |
| Minnesota | Industrial Commission. | Compulsory | Board of 3 appointed by commission. Medical findings conclusive.[4] | 3 years after last exposure, last payment, or default. | 3 years after last exposure. | Disability payments; award prorated for aggravation. | Unlimited | Not compensated unless following a compensable period of total disability. | $500 maximum first month act effective (April 1943); increased by $50 each month until maximum is same as for accidents. 25 weeks' maximum benefits for employee removed from employment because of undue hazard. Partial waiver for nondisabled silicotics. | 5 years' exposure in last 10 with 3 years in State |
| Missouri[1] | Workmen's Compensation Commission. | Elective | No provision | 1 year after injury, death, or last payment. | 300 weeks after injury. | Disability payments. | Limited to 90 days and $750; may be extended. | Compensated. | No provision | No provision. |

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|---|---|---|---|---|---|---|---|---|---|---|
| Montana | State Department of Public Welfare. | (6) | do | No provision | No provision | No provision | No provision | Not compensated. | $30 per month for total disability (payable from public funds.) | Do. |
| Nebraska[1] | Workmen's Compensation Court. | Elective | do | 6 months after injury or death; barred unless filed in 1 year after injury, death, or last payment. | 325 weeks after injury. | Disability payments; award prorated for aggravation. | Unlimited | Compensated | No provision | Disability within 2 years of last exposure. |
| New Jersey | Workmen's Compensation Bureau in Department of Labor. | do | do | 1 year after last exposure, last payment or default. | 3 years after last exposure or 7 years if following continuous disability beginning within 3 years. | do | Limited to $50 medical and $50 hospital; may be extended. | Not compensated. | $1,000 maximum first month act effective (January 1945); increased by $50 each month until maximum is same as for accidents. | 5 years' exposure in last 10; 1 of 5 in State.[3] |
| New Mexico | Courts | do.[7] | do | 1 year after disablement or 6 months after death. | 2 years after last exposure or 5 years if following continuous disability. | Disability payments; award prorated for aggravation. | $400 limit | Compensated | $800 maximum first month act effective (April 1943); increased by $70 each month until maximum is $5,000. Payable at rate of $15 per week, plus $1.50 for each dependent child in death cases. | Exposure for 1,250 work shifts in State during last 10 years. |
| New York | Workmen's Compensation Board. | Compulsory | Board of 3 appointed by board; Report prima facie evidence of medical facts. | 1 year after injury or death; board may extend to 2 years. | 5 years after last exposure and following continuous disability. | Disability payments. | 90-day limit; may be extended to 450 days. | Not compensated. | Maximum for temporary total disability is $5,000 and for permanent total disability and death $7,500. | Disability within 1 year after last injurious exposure. |
| North Carolina | Industrial Commission. | Elective | Board of 3 appointed by the Commission; medical findings advisory. | 1 year after disablement or death; 2 years after last payment.[8] | 3 years after last exposure or 7 years if following continuous disability beginning within 3 years.[8] | do | 3 years; not exceeding $1,000 a year. | Compensated | 40 weeks' maximum benefit for nondisabled silicotics removed from employment; $500 maximum for special training; partial waiver permitted. Rate reduced in cases complicated with tuberculosis. | 2 years' exposure within State during last 10 years. |
| North Dakota[1] | Workmen's Compensation Bureau. | Compulsory | No provision | 60 days after injury or death; barred after 1 year. | 6 years after injury. | Award prorated for aggravation. | Unlimited | Compensated | None | No provision. |

See footnotes at end of chart.

## WORKMEN'S COMPENSATION—SILICOSIS

### Summary of Statutory Provisions—June 1, 1946

*Chart IV—Cont'd*

| State | Administrative agency | Application of law | Medical boards | Time limit on claim filing | Time limitation on eligibility for death benefits | Deductions from death awards | Medical care | Partial disability | Special liability limitations | Requirements for exposure |
|---|---|---|---|---|---|---|---|---|---|---|
| Ohio | Industrial Commission. | do | 3 silicosis referees appointed by commission; findings advisory; also, medical boards of review; medical findings conclusive if favorable to claimant. | 1 year after disablement or 6 months after diagnosis of silicosis by physician; 6 months after death. | 8 years after last injurious exposure, but if death followed continuous total disability commencing within 8 years after such exposure, requirement of death within 8 years shall not apply. | Disability payments, when total of disability payments and death award amounts to $7,500. | $200 limit; may be increased by unanimous approval of commission. | Not compensated; but 26 weeks maximum benefits allowed for nondisabled silicotics removed from employment. | do | 3 years' exposure within State. |
| Oregon | Industrial Accident Commission. | Elective | Medical board of review for rejected claims; medical decision final. | do | 2 years after last injurious exposure. Limitation, however, not applicable if continuous total disability commencing within the 2-year period. | | $250 limit; may be increased. | Compensated. Amount of award determined by condition and considering statutory limitation. | If exposure in State less than 5 years after July 1, 1943, $500 maximum first month act effective (July 1943); increased by $50 each month until maximum is $3,000. If more than 5 years' exposure in State after July 1, 1943, compensation same as for accidents. | 5 years' exposure in State, part after effective date of act. |
| Pennsylvania | Workmen's Compensation Bureau in Department of Labor and Industry. | Elective | Board of 3 appointed by secretary of labor and industry; medical findings conclusive. | 1 year after disablement, death, or last payment. | 3 years from date of last employment. | Disability payments. | Medical, 60 days and $150; hospital, 60 days; may be extended 30 days, maximum $75. | Not compensated. | Maximum benefits $4,000 employer's liability increasing from ½ in 1939 to full amount in 1949; State paying difference from special appropriations. | 4 years' exposure within State in last 8 years. |
| Puerto Rico | State Insurance Fund. | Compulsory | No provision | No provision | 2 years after injury. | No provision | Unlimited | Compensated | None | Disability within 1 year after last exposure. |
| Rhode Island | Division of Workmen's Compensation in Department of Labor. | Elective | do | 2 years after injury or death. | 3 years after last exposure or 7 years if following continuous disability beginning within 3 years. | Disability payments; award prorated for aggravation. | 90-day limit; may be extended to 180 days. | do | $500 maximum first month act effective (October 1942); increased by $50 each month until maximum is $5,000. Partial waiver for nondisabled silicotics. | Disability within 3 years after last injurious exposure. |
| Utah | Industrial Commission. | Compulsory | do | 1 year after cause of action arises; death cases 6 months. | 2 years from last working day exposed, or 5 years if following continuous disability. | do | Limited to $500. | do | $3,000 maximum if disability or death occurs in July 1945; increased by $50 each month until maximum is $5,000. | 5 years' exposure within State in last 10 years. |

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|---|---|---|---|---|---|---|---|---|---|---|
| Virginia[1] | do | Elective | do | 1 year after disability or death. | 6 years after injury. | Disability payments. | Limited to 60 days; may be extended 180 additional days. | do | None | No provision. |
| Washington[1] | Department of Labor and Industries. | Compulsory | do | (9) | No provision | No provision | Limited to compensable period; may be extended. | do | do | Do. |
| West Virginia | Workmen's Compensation Commissioner. | Elective | Board of 3 appointed by Commissioner; medical findings conclusive unless objected to within 15 days. | 1 year after last exposure or 6 months after death. | 6 years after last exposure. | No provision | None | Compensated; maximum for first stage $800; second stage $1,600. | For total disability (known as "third stage") compensation same as for accidents. Payment for partial disability (first or second stage) constitutes waiver of further compensation rights. | 2 years' exposure in same employment within State. |
| Wisconsin | Industrial Commission. | Compulsory | No provision | 2 years from injury or death, or from date claimant knew or should have known of disability and relation to employment. | No provision | do | Unlimited | Compensated | $3,500 maximum for "nondisabled silicotics"; 90 days' exposure required. | No provision. |
| United States: | | | | | | | | | | |
| Civil Employees[1] | U. S. Employees' Compensation Commission. | do | do | Disability: 60 days after injury; may be extended to one year. Death: 1 year after death. | do | do | do | | None | Do. |
| Longshoremen[1] | do | do | do | 1 year after injury, death or last payment. | do | do | do | do | do | Do. |

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[1] The provisions for silicosis do not differ from provisions for other occupational diseases.

[2] "Conclusive evidence" in favor of the claim is necessary if the required exposure has not been experienced.

[3] These provisions apply only in granite industry. Silicosis, other than that contracted in this industry, is subject to the same provisions as other occupational diseases.

[4] This provision has been held unconstitutional.

[5] For rights accruing prior to May 28, 1945, benefits are increased at the rate of $50 a month, and aggregate amount is $4,000.

[6] Pension payable to any person who has been a resident of State for 10 years immediately preceding application.

[7] Applies to employers of 4 or more, except agricultural laborers and domestic servants.

[8] Applicable to lead poisoning also.

[9] By court decision limitation on claim filing is not applicable to occupational diseases.

**U. S. DEPARTMENT OF LABOR**
**Division of Labor Standards**

## WORKMEN'S COMPENSATION—MEDICAL BENEFITS
### Summary of Statutory Provisions—June 1, 1946

***Chart V***

| State | Limitations by statute | | Extensions permitted | | Artificial appliances furnished | Other modifications |
|---|---|---|---|---|---|---|
| | Period | Amount | Period | Amount | | |
| Alabama | 90 days | $200 | | | Crutches and apparatus | |
| Alaska | 1 year | Unlimited | | | do | |
| Arizona | 100 months | do | | | do | Additional medical aid may be furnished by the rehabilitation fund. In case of occupational disease, benefits are payable only for total disability, maximum $500. |
| Arkansas | 60 days | do | Unlimited | | do | 90-day limit in case of silicosis; may be extended to 180 days. |
| California | Unlimited | do | | | Crutches and apparatus including artificial members. | |
| Colorado | 4 months | $500 | | | Crutches, apparatus, artificial members and glasses (1) | |
| Connecticut | Unlimited | Unlimited | | | | |
| Delaware | 30 days | $200 | Unlimited | Unlimited | No provision. | |
| District of Columbia | Unlimited | Unlimited | | | Crutches and apparatus. | |
| Florida | Unlimited | $1,000 | | Unlimited | Crutches, artificial members and apparatus. | |
| Georgia | 10 weeks | $500 | Unlimited [2] | | Artifiicial members. | |
| Hawaii | Unlimited | Unlimited | | | Artifiicial members and glasses. | |
| Idaho | do | do | | | Crutches and apparatus | 90-day limit in case of occupational diseases; may be extended for an additional 90 days. |
| Illinois | do | do | | | Artificial members and braces | 6-month limit in case of silicosis or asbestosis. |
| Indiana | 120 days | do | | | do | 30-day limit in case of occupational diseases; may be extended for 30 additional days. |
| Iowa | Unlimited | $600 | | | No provision | |
| Kansas | 60 days | $100 | | $500 | Crutches and apparatus | |
| Kentucky | 90 days | $200 | Unlimited | $300 | Appliances | |
| Louisiana | Unlimited | $500 | | | No provision | |
| Maine | 30 days | $100 | Unlimited | Unlimited | Mechanical and surgical aids | |
| Maryland | Unlimited | $750 | | | Crutches, apparatus and artificial members. | Additional benefits of not more than $250 for hospital services. |
| Massachusetts | do | Unlimited | | | Artificial eye or limb or other mechanical appliance. | |
| Michigan | 6 months | do | 6 months | | Crutches, artificial limbs, eyes and teeth, eye glasses, hearing apparatus and other necessary appliances. | |
| Minnesota | Unlimited | do | | | Crutches and apparatus including artificial members. | |
| Missouri | 90 days | $750 | Unlimited | Unlimited | No provision | |
| Montana | 9 months | $500 | | | Artificial arm, hand, leg, foot, eye, or teeth. | |
| Nebraska | Unlimited | Unlimited | | | No provision | |
| Nevada | 6 months | do | 1 year | | do | |
| New Hampshire | 90 days | do | | | Artificial eye, limb, or other member | |

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| New Jersey | Unlimited | Hospital, $50; medical, $50. | | Unlimited | Artificial limb or other appliance including artificial teeth or glass eye. | |
| New Mexico | do | $400 | | | Artificial members includnig teeth and eyes. | |
| New York | do | Unlimited | | | Crutches and apparatus | In case of dust disease 90-day limit; may be extended for 360 additional days. |
| North Carolina | 10 weeks | Unlimited | Unlimited | | Artificial members | Unlimited for occupational diseases but limited to 3 years, maximum $1,000 per year for silicosis or asbestosis. |
| North Dakota | Unlimited | do | | | Artificial limbs, glasses, braces, or appliances. | |
| Ohio | do | $200 | | Unlimited | Artificial appliances | Extension subject to unanimous approval of commission. |
| Oklahoma | 60 days | Unlimited | Unlimited | | Crutches and apparatus | |
| Oregon | Unlimited | Hospital, $100; medical, $100; sundry, $50. | | Unlimited | Artificial leg or arm[3] | |
| Pennsylvania | 60 days | $150 | 30 days | $75 | No provision | Also hospital services for 60 days, maximum $150; may be extended for 30 days; maximum $75. |
| Puerto Rico | Unlimited | Unlimited | | | do | |
| Rhode Island | do | $300, or $500 if hospital period of over 14 days. | | Unlimited | Eye glasses, dentures, braces and supports, artificial limbs and crutches and other similar appliances. | 90-day limit for silicosis or asbestosis; may be extended for 90 days. |
| South Carolina | 10 weeks | Unlimited | Unlimited | | Artificial members | |
| South Dakota | 20 weeks | Hospital, $200; medical $200. | | | Apparatus | Additional benefits of $500 may be allowed State employee. |
| Tennessee | 3 months | Hospital, $100; medical, $100. | | Hospital, $100; medical, $300. | do | |
| Texas | 4 weeks | Unlimited | To 91 days | | Artificial appliances | Hospital services may be extended for 2 additional weeks. |
| Utah | Unlimited | $500 | | Unlimited | do | In case of occupational disease, benefits payable only for total disability, maximum, $500. |
| Vermont | Hospital, 60 days; medical, 60 days. | Hospital, $300; medical, $75. | | | No provision | Unexpended balance of $75 for medical services may be applied to hospital expenses. |
| Virginia | 60 days | Unlimited | 180 days | | do | |
| Washington | Unlimited | do | | | Artificial members | In temporary disability benefits limited to period of compensation. |
| West Virginia | do | $800 | | $800 | Crutches, artificial limbs and other mechanical appliances. | Also $800 may be allowed for vocational rehabilitation. No medical benefits for silicosis. |
| Wisconsin | do | Unlimited | | | Crutches, artificial members and appliances. | |
| Wyoming | do | Hospital, $165; medical, $165. | | Hospital, $330; medical, $220. | Artificial members, hearing aids, and other similar braces. | Further hospitalization may be authorized by court for 6-month periods. |
| United States: | | | | | | |
| Civil employees | do | Unlimited | | | Appliances[4] | |
| Longshoremen | do | do | | | Crutches and apparatus | |

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[1] By court decision, artificial appliances are required to be furnished.

[2] Additional benefits for such time as will lessen period of disability.

[3] Remains property of State.

[4] Orthopedic or prosthetic appliances may be furnished if likely to cure or give relief or reduce the degree or period of disability.

**U. S. DEPARTMENT OF LABOR**
**Division of Labor Standards**

# WORKMEN'S COMPENSATION—SECOND-INJURY FUNDS

## Summary of Provisions in States Having Provided Second-Injury Funds or Equivalent Arrangements—June 1, 1946

***Chart VI***

| State | Injuries covered | Payable by employer | Payable by fund | Source of fund | Special provisions |
|---|---|---|---|---|---|
| Alaska | Second injury resulting in permanent partial disability which, added to preexisting injury, results in permanent total disability. | Disability caused by second injury. | Difference between compensation payable for second injury and permanent total disability. | $1,500 in no-dependency death cases; 60% of amount payable to nonresident alien dependents (other than wives and children); 40% of amount payable to wives and children who are nonresident aliens; 2% of compensation payable in case of specific injuries. | |
| Arizona | Second injury involving loss or loss of use of member or eye which, added to pre existing loss or loss of use of member, results in permanent total disability. | do | do | $1,150 in no-dependency death cases. | Payments are also made from fund for vocational rehabilitation. |
| Arkansas | Second injury involving loss of member or eye, which, added to pre existing loss of member, results in permanent total disability. | do | do | $500 in no-dependency death cases. | Employer liable for combined disability if both injuries in same employment. |
| California | Second injury resulting in permanent partial disability, which, added to preexisting disability resulting from loss or loss of use of member or eye, results in 70 percent or more of permanent total disability. | do | do | Legislative appropriation of $200,000. | Payments are made by State Treasurer. |
| Colorado | Second injury involving loss or loss of use of member or eye, which, added to preexisting loss or loss of use of member, results in permanent total disability. | do | do | $500 in no-dependency death cases. | Amount payable by fund is limited to ½ of average wage loss, in case employee obtains employment while receiving benefits. |
| Connecticut | Second injury involving loss or loss of use of member or eye, which, added to preexisting loss or loss of use of member or eye incurred by "accidental injury, disease, or congenital cause," results in permanent total disability. Also covers reduction of sight in 1 eye to 1/10 or less of normal vision, followed by similar reduction in sight in other eye. | do | do | 1 percent of compensation paid by insurance carriers and self-insurers during preceding calendar year. | Payments suspended when fund amounts to $50,000 and resumed when fund is reduced below $50,000. |
| Delaware | Second injury which, added to preexisting permanent injury "from any cause," results in permanent total disability. | do | do | Tax of ½ of 1 percent on premium received by insurance carriers and an equivalent charge on self-insurers. | Payments to fund are suspended when it amounts to $30,000 and resumed when fund is reduced below $20,000. |

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| District of Columbia | Second injury resulting in permanent partial disability, which, added to preexisting injury results in permanent total disability. | do | do | $1,000 in no-dependency death cases. | 50 percent of fund used to pay additional benefits in second-injury cases and the balance for vocational rehabilitation. |
| Hawaii | Loss of hand or foot, followed by loss of another hand or foot, or loss of eye followed by loss of other eye. | do | do | $500 in no-dependency death cases. | |
| Idaho | do | do | do | Amount equal to 2 percent of award for schedule injuries. | |
| Illinois | Second injury involving loss or loss of use of member or eye, which, added to preexisting loss or loss of use of member, results in permanent total disability. | Disability caused by the second injury. | Difference between compensation payable for second injury and permanent total disability. | $400 in no-dependency death cases; $100 in case of loss of member. | When fund amounts to $50,000 in excess of expenditures to be made in the ensuing 6 months, commission shall reduce or suspend contributions in case of loss of member. |
| Iowa | do | do | Difference between compensation payable for second injury and permanent total disability, less amount paid for previously lost member or organ. | $100 in death cases; sums contributed by the United States; payments due but not paid to nonresident alien dependents; and sums recovered from third party. | Payments suspended when fund amounts to $50,000 and resumed when fund is reduced below $30,000. |
| Kansas | do | do | do | $500 in no-dependency death cases and appropriation of $25,000. | Payments suspended when fund amounts to $50,000 and resumed when fund is reduced to $25,000. |
| Kentucky | Second injury which, when added to preexisting permanent partial disability results in disability greater than from second injury alone.[1] | do | Difference between compensation payable for second injury and permanent total disability less amount paid for prior injury. | Tax of ¾ of 1% on premium received by insurance carriers and a similar tax on self-insurers. | Payment suspended when amount in fund exceeds $75,000 as of June 30 of any year. |
| Maine | Second injury involving loss or loss of use of member or eye which, added to preexisting loss or loss of use of member, results in permanent total disability. | do | Difference between compensation payable for second injury and permanent total disability. | $300 in no-dependency death cases. | |
| Maryland | do | do | do | $100 in death cases; $10 in each case of injury causing permanent partial disability; and sums contributed by United States. | Payments suspended when fund amounts to $100,000 and resumed when fund is reduced to $50,000. |
| Massachusetts | do | ½ of amount due for permanent total disability or death, plus amount for schedule injuries. | ½ of amount due for permanent total disability or death. | $500 in no-dependency death cases. | |
| Michigan | Second injury involving loss of member or eye, which, added to preexisting loss of member, results in permanent total disability. | Disability caused by the second injury. | Difference between compensation payable for second injury and permanent total disability, less amount previously paid for both injuries. | $1,000 in no-dependency death cases, less previous payment for disability. | |
| Minnesota | Second injury resulting in permanent partial disability, which, added to preexisting disability, results in permanent total disability. | do | Difference between compensation payable for second injury and permanent total disability. | $300 in no-dependency death cases; 2 percent of award in permanent partial disability cases. | Payments are also made from fund for additional benefits in cases of permanent total disability and death. |
| Missouri | do | do | do | $500 in no-dependency cases; $100 in case of total loss of use of leg, arm, hand, or eye. | When fund exceeds $50,000 commission shall reduce or suspend contributions. |

See footnotes at end of chart.

**U. S. DEPARTMENT OF LABOR**
**Division of Labor Standards**

## WORKMEN'S COMPENSATION—SECOND-INJURY FUNDS

### Summary of Provisions in States Having Provided Second-Injury Funds or Equivalent Arrangements—June 1, 1946

*Chart VI—Cont'd*

| State | Injuries covered | Payable by employer | Payable by fund | Source of fund | Special provisions |
|---|---|---|---|---|---|
| New Jersey | do | do | do | 1 percent of losses for previous years by carriers and self-insurers. | Contributions cease whenever the fund attains $1,500,000. |
| New York | Second injury involving loss or loss of use of member or eye, which, added to preexisting injury, results in permanent total disability; or second injury, which, added to preexisting injury involving loss or partial loss of member or eye or other "permanent physical impairment" not resulting from a dust disease, results in disability greater than from second injury alone. | Benefits for first 104 weeks of disability. | Compensation payable for disability after first 104 weeks. | Assessment against insurance carriers on a basis proportionate to the total compensation payments made by all carriers. | If subsequent injury or disease results in death, and injury or death resulted because of preexisting disability, employer is liable for death benefits for first 104 weeks, and the special fund is liable for death benefits thereafter. In all cases, the employer or insurance carrier is required to pay awards and medical expenses, but is reimbursed from the special disability fund for benefits subsequent to those payable for the first 104 weeks. |
| North Carolina | Second injury involving loss of member or eye, which, added to preexisting loss of member, results in permanent total disability, provided that original and increased disability were each 20 percent of the entire member. | Disability caused by the second injury. | Difference between compensation payable for second injury and permanent total disability. | Commuted amount in death cases if no dependents or next of kin; ½ of that amount when next of kin; $100 in each case of 50 percent or more of loss or loss or use of major member; $25 in each case of loss or loss of use of minor member. | |
| North Dakota | Second injury which, when added to preexisting injury, results in permanent total disability. | do | | | Compensation in excess of amount chargeable to second injury is charged to surplus fund. |
| Ohio | Second injury involving loss of member or eye, which, added to preexisting loss of member, results in permanent total disability. | do | | | In the case of a self-insuring employer, excess payments are made from the surplus fund. By rule of the commission, in the case of State fund employer, compensation in excess of amount chargeable to second injury is charged to surplus fund. |
| Oklahoma | Second injury to "physically impaired person" resulting in additional permanent disability greater than results from second injury alone. "Physically impaired person" is defined as one having a permanent partial disability due to accident, disease, birth, military action, or other cause. | do | Difference between compensation payable for second injury and permanent total or permanent partial disability. In the case of a permanent partial disability, the percentage of disability due to the employee's condition as a "physically impaired" employee is deducted from award. | 1 percent of losses for previous year by carriers and self-insurers, and 1 percent of awards for permanent partial disability by employees. | Payments suspended when fund amounts to $100,000 and resumed when fund is reduced below $50,000. |
| Oregon | Second injury involving loss of member or eye, which, added to preexisting loss of member, results in permanent total disability. | do.[2] | Difference between compensation payable for second injury and permanent total disability. | Transfer of $250,000 from industrial accident fund, and monthly transfer of 2½ percent of total receipts of accident fund. | Industrial accident commission may pay compensation benefits from second injury fund if the industrial accident fund becomes exhausted. When unexpended balance in fund is greater than $200,000, the percentage of receipts to be transferred to the fund is reduced or suspended. |

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| Pennsylvania | Second injury involving loss or loss of use of member or eye, which, added to preexisting loss or loss of use of member, results in permanent total disability. | Disability caused by the second injury. | Difference between compensation payable for second injury and permanent total disability. | Legislative appropriation of $100,000. | Payments are made directly by the State. |
| Puerto Rico | Second injury, which, added to preexisting injury, results in permanent disability. | do | | | Compensation in excess of amount chargeable to second injury is paid by the reserve fund for disasters. |
| Rhode Island | Second injury involving loss or loss of use of member or eye or any definite permanent partial disability, which, added to preexisting loss or loss of use of member, results in permanent total disability. | do | Difference between compensation payable for second injury and permanent total disability. | 1 percent of gross premiums collected by insurers and 1 percent of premiums self-insurer would have had to pay to obtain insurance, and $750 in no-dependency death cases. | |
| South Carolina | Second injury involving loss of member or eye, which, added to preexisting loss of member, results in permanent total disability. | do | do | Commuted amount in death cases if no dependents or next of kin; ½ that amount when next of kin. | |
| Tennessee | Second injury involving loss or loss of use of member or eye, which, added to preexisting loss or loss of use of member, results in permanent total disability. | do | do | $100 in death cases and $10 in each case of permanent partial disability. | |
| Utah | Second injury which, when added to preexisting injury, results in disability greater than from second injury alone. | do | Difference between compensation payable for second injury and compensation for combined injuries. | In no-dependency death cases, 20 percent of benefits provided for total dependents. | Payments are also made from fund for vocational rehabilitation, maximum $520. |
| Washington | Second injury or disease, which, when added to preexisting injury or disease, results in permanent total disability. | do.[2] | Difference between charge assessed against employer at time of second injury and total cost of pension reserve. | Transfer of not more than $500,000 from accident fund to seccond-injury fund. | |
| West Virginia | Second injury, which, added to preexisting loss or loss of use of member or eye, results in permanent total disability. | do | Difference between compensation payable for second injury and permanent total disability. | | Compensation in excess of amount chargeable to second injury is paid out of any funds at the commissioner's disposal. |
| Wisconsin | Second injury consisting of 15 percent or more of permanent total disability provided preexisting injury was also 15 percent or more of permanent total disability. | do | Disability caused by lesser of 2 injuries. | $75 in case of loss of member, ear, or eye. | If the combined disabilities result in permanent total disability, the amount payable by the fund is the difference between the compensation payable for the second injury and permanent total disability. |
| Wyoming | Second injury in extra-hazardous employment, which, added to preexisting loss or loss of use of member or eye, results in permanent total disability. | do | Difference between compensation payable for second injury and permanent total disability. | $500 in no-dependency death cases; appropriation by legislature; and sums contributed by the United States. | Any payments for previous permanent partial disability or payments which would have been made if the prior injury had occurred in an extra-hazardous employment are deducted from the award. |
| United States:<br>Longshoremen | Second injury resulting in permanent partial disability, which, added to preexisting injury, results in permanent total disability. | do | do | $1,000 in no-dependency death case. | 50 percent of fund used to pay additional benefits in second injury cases and the balance for vocational rehabilitation. |

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[1] Does not apply to diseases except when contracted by the employee while a member of the armed forces of the United States and sustained in line of duty while the United States was engaged in war.

[2] Employer is liable for "experience rating purposes." Actually all payments are made by the industrial accident fund.

[3] Benefits are paid by the accident fund, but the accident cost rate of the employer at the time of the second injury is charged only with the accident cost which would have resulted solely from the second injury.

# CHAPTER III

# THE NEED OF SCIENTIFIC PROOF IN MEDICOLEGAL CONTROVERSY

The lack of mutual interdependence between law and medicine is not peculiar to the administration of workmen's compensation, for we find it to exist in all our various courts of justice. Leaders in the legal profession are greatly concerned with the inadequacy of our present trial procedures and are demanding a better mechanism which would bring *scientific evidence to the point of maximum legal utility.* Professor Roscoe Pound[1] proposed the creation of a Ministry of Justice as a means of "effecting rapid and successful transfusion of scientific thought into the circulating blood stream of law." Professor Hubert W. Smith[2] suggests the formation of a National Scientific Commission to serve as a master censor for the courts. "Such a body, made up of qualified legal and scientific persons, could probe into the merits of each species of scientific proof and lay down appropriate criteria, safeguards and cross checks needed to make testimony trustworthy. . . . . Such a body could develop also a comprehensive system for certifying the proficiency of witnesses."

If the law in general is aware of the fact that much of the testimony in our common courts is unreliable, it is high time that the authorities responsible for the equitable administration of workmen's compensation also recognize this obvious truth. It is an undeniable fact that the bulk of medical testimony given before the Industrial Accident Commission is *conjectural.* That unqualified medical testimony of this sort should have been deemed acceptable throughout the thirty-six years that the compensation act has been in existence is the fault of law and medicine combined. Redress of this wrong is more easily attained in workmen's compensation than in other forms of legal controversy, since the decision usually resides in the hands of one person whose experience and training should enable him to evaluate facts of a scientific nature, and to differentiate these facts from conjecture. But such insight is not to be expected of lay juries. If justice is to prevail in the administration of workmen's compensation, there will have to be established a fact-finding mechanism consisting of a program of adherence to scientific proof; a structure "horse high, pig tight and bull strong." [3]

The author's concern in this need for qualified medical testimony extends beyond the desire to see workman or employer accorded justice; it stems from the desire to have accepted occupational disease entities established and maintained. The direct traumas of industry seldom lack scientific proof, since the cause and evidence of injury are so apparent as not to require further testimony to the fact. When such cases are brought before the commission, it is usually in order to determine the presence and/or the degree of disability of the injured. But it is in the alleged or actual occupational disease that there arises the necessity for scientific proof. Granting that medicine is not an exact science, and that clinical judgment and with it the diagnosis is bound to vary, there exists, nevertheless, a limit to justifiable differences, and no physician should be permitted to express glib opinions for which he can offer no accepted or established proof.

If a group of physicians were picked at random from any given community, all of these, or practically all, could outline the basic tenets for the diagnosis of tuberculosis, pneumonia, malaria, or kidney stone. The reason why there exists uniformity of diagnosis with regard to the communal diseases may be found in the fact that medical school instruction and subsequent experience have provided the average physician with adequate knowledge on these subjects. Occupational diseases, on the other hand, are not taught in our medical schools, and only in rare instances will a subsequent practice bring the average physician into intimate contact with industry or industrial medicine.

According to a questionnaire sent out several years ago by the American Medical Association, it was concluded that out of 120,000 physicians only 1,283 could be recorded as competent to practice industrial medicine. The expansion of medical programs throughout industry has of course increased the number of trained industrial physicians, although there are still far too few. This is one of the reasons why medical testimony in compensation controversies is so very often unqualified. As yet, however, this obvious truth has not impressed our compensation courts sufficiently to create the demand for a more efficient system.

No referee fully cognizant of established legal tenets would permit the application of defective principles of law to agreed facts, but the same referee will blindly ignore the distortion or abuse of medically acceptable facts. Decisions based on scientifically inadequate proof become precedents for subsequent rulings. If this abuse is continued, it will greatly hinder the teaching of occupational disease entities and the advancement of industrial medicine. It would be comparatively simple to establish instead the adherence to medically scientific facts if our legal friends would evaluate the quality of medical testimony and discredit conjecture.

In discussing this problem, many referees contend that it is not always possible for them to differentiate between a qualified and an unqualified medical witness. They argue that it is the responsibility of the medical profession to establish this qualification; that the state or county medical societies should provide a panel of men who, by training and experience, could be considered competent medical experts in problems of industrial diseases. In this respect, a plan similar to the one in force in Minnesota deserves consideration. In 1943 the act in that state provided for the appointment of a medical review board composed of three doctors of medicine selected from a panel of fifteen nominees, to be chosen by the Dean of the College of Medicine of the University of Minnesota, the Council of the Minnesota State Medical Society, and the Governor of Minnesota. Ten nominees must be doctors of medicine with at least five years experience in the diagnosis, treatment, and care of industrial diseases, while five others must be doctors of medicine with at least five years of specialization in the field of x-ray diagnosis and treatment. One member of the board is to be selected by each party to the controversy, and the two doctors so selected will choose a third member. This plan appears to be a reasonable approach to the problem.

Some interested individuals who seek to correct the existing abuse of scientific proof have suggested that the county medical societies discipline any member who testifies falsely or ignores scientific proof in his testimony. But these proponents forget that under existing conditions many physicians are not members of their local medical society and that to these discipline would be no threat. But regardless of the method of reform chosen, the need and the opportunity are one.

### References

1. Symposium on Scientific Proof and Relations of Law and Medicine. Clinics, Philadelphia, 1943, J. B. Lippincott Co., vol. I, no. 6.
2. Ibid.
3. Ibid.

# CHAPTER IV

# FUNCTIONS OF THE INDUSTRIAL PHYSICIAN

*Medicine in industry is no longer limited to medicine per se—it is a varied and extensive service.* The present-day concept of the duties of an industrial physician is in sharp contrast with (1) the industrial surgeon, who confined his interest to the traumas of industry; (2) the "part-time" physician, who spent an hour or two each day or specified days at a plant; (3) the "on-call" physician, who conducted a general practice in the neighborhood of the plant and to whom were sent the emergency cases; and (4) the "uptown" surgeon, to whom the insurance company or the employer referred the seriously injured workman. Most of these physicians never saw the inside of the plant which employed their services.

Ideally, the industrial physician is one who has had undergraduate and postgraduate training in this type of medicine, has served an in-plant internship and/or a year or two of apprenticeship in industry. With such a background he should have a fundamental knowledge of industrial hygiene and toxicology. Such, indeed, would be the ideal. But since industry is demanding medical care faster than medical education can supply adequately trained physicians, it is obvious that physicians self-educated in matters of industrial health must satisfy the present need. To their information this book is dedicated. In addition to describing the ideal medical service in industry, this chapter will also attempt to acquaint the general practitioner with certain obligations he must fulfill in dealing with an industriallv ill or injured workman.

The responsibilities of the full-time industrial physician vary with the size of his organization and the type of work engaged in. Obviously, a medical program necessary for a heavy industry or one where toxic chemicals are in use entails features not required for a department store. The following description of a good medical service must therefore be adjusted to meet the specific requirements of each particular plant, retaining the essential purpose of preventive medicine. To do this, some or all of the following services should be established:

1. *The maintenance of one or more emergency units.* These should be located in accessible places but not in the neighborhood of excessive noise, vibrations, odors, or other annoying factors. If a sufficient number of these units are not provided or are not readily accessible, the workmen are prone to neglect minor injuries which may develop into major problems.

Although the majority of these units are set up to function for the purpose of first aid only, the central unit should be equipped as an all-purpose medical department. Optimum service demands several examining rooms or cubicles with adjoining dressing rooms, one or more operating rooms for minor and major surgery, x-rays, and physiotherapy, a laboratory, record files, and equipment for conducting special examinations of the eyes, ears, etc. The area allotted to the central unit should be spacious, well planned to handle a maximum number of patients, and at the same time offer efficient care.

2. *The pre-employment examination.* In addition to a complete physical examination, the routine should include blood count, urine analysis, Wassermann test, and chest x-rays. But it is no longer good practice to make all applicants fulfill the same requirements or to limit the requirements to all applicants. The pre-employment examination provides the basic information for the proper placement of all employees. In the applicant it must consider age, sex, physical handicaps, previous general health, previous occupational diseases, previous type of employment, and so on; in placing the individual, job requirements must be considered, such as climbing, lifting, crawling, temperature levels, toxic chemicals, dusts, etc. For the sake of illustration: an applicant with a history of previous liver damage should never be assigned to work which exposes him to the hydrocarbons; an individual with epilepsy to high levels; or an individual with asthma to dusty areas.

One of the most commendable advancements in industrial medicine has been the emphasis upon the importance of this pre-employment examination, not only as to its thoroughness, but also as to the competence of the examiner. This is no longer an assignment for the misfit physician who has failed to succeed in general medicine, or for one who, because of age, questionable habits, or indifference, has lost contact with good medical practice. It is now held that the physician conducting the pre-employment and the periodic health examinations should be a good clinician; that he should know his plant as he knows his own home; and that he should understand the hazards inherent in all the processes which are carried on in the plant.

3. *The periodic re-examination.* Here, again, the required frequency of examination and its extent depend largely upon the occupation of the individual workman or group of workmen. Assuming that all employees are examined annually or semiannually, certain employees will need more frequent checkups, as, for instance, those exposed to lead or to benzol or to certain of the hydrocarbons. In some trades, the workmen should be made to present their individual protective equipment for careful periodic inspection as to its efficiency. Such pre-employment and periodic examinations constitute a preventive bulwark against disease and will prove the best means of maintaining optimum adult health throughout the country.

No adequate health program in industry will excuse management and those in executive positions from submitting to pre-employment and periodic examinations. It is not unusual to find an industrial organization compelling its laborers to undergo physical examinations but not requiring the office force to do so. Too often an important executive, working hard and long hours, suddenly drops out of a key position because of a coronary accident. It stands to reason that his physical status should be known and his habits of work controlled, just as those of the man employed to operate a degreasing machine. Preventive medicine in industry should recognize no line of demarcation at the front office.

4. *The placement program.* This is an integral part of good industrial practice. The object of physical examination is not to reject the applicant with anything less than perfect health but to place him where he may make use of his capabilities. A full discussion of this subject will be found in a subsequent chapter.

5. *Frequent plant surveys.* A good medical director will look upon his plant as a good housewife does upon her home and will do justice to the fact that it needs periodic cleaning from top to bottom. Plant housekeeping requires constant vigilance over the safety program, the efficiency of protective procedures such as ventilation, and certain individual equipment as well as the secure functioning of exhausts, and illumination with special precautions against potential fire hazards. In various ways the workmen must be induced to cooperate fully by using the protective equipment provided to them. Many cases of occupational disease are traceable to negligence. It should also be kept in mind that exhaust systems or ventilators are subject to frequent failures which can be detected only by regularly repeated checking.

6. *Cooperation with related personnel.* Plant safety and plant hygiene are the responsibility of the medical director. Frequent discussion periods with those in charge elevate the standard of injury prevention. Such meetings should include the industrial relations director, for with his department medicine has an allied function. *Good medicine is a lubricant between management and labor.*

7. *The educational program.* Industrial health includes programs devoted to nutrition, dental and mental hygiene, in-plant and out-plant nursing care, social activities, sports, and theatrical entertainment. While some of these may not be the direct responsibility of the medical director, he should support and encourage such activities. During the frenzy of war production, management discovered that output was increased by the introduction of a variety of activities which a few years previously would have been considered lunacy. The pre-war industrialist would have prohibited music in his plant, rest periods, coffee and doughnuts at the end of the aisleways, etc. With the war

over, most plants are continuing these various innovations. In the weekly newspaper published more than one year after the end of the war by one of the West Coast airplane companies, one reads: "Lunchbox Follies Beckons Thespians." Following this headline it states: "There will be nothing usual or ordinary about the lunch period for the next several months . . . . . for the Plant One Stage and the Crenshaw Stage will rock to the efforts of the N.R.C. entertainers." The article goes on to invite all the members of each shift to compete for prizes. In this same company newspaper there is a column entitled "N.R.C. Good Time Table" and underneath are listed the following activities: Art Club, Baseball, Badminton, Softball, Flying Club Association, Sea Scouts, Swimming Club, Volleyball, Theatricals, Horse Shoe Club, etc. Surely such a program within industry will do much to make men want to work. Someone has pointed out that there is a great deal of work in pleasure—that the golfer, bowler, hunter, or fisherman works hard for the pleasure he derives. Conversely there can be *a great deal of pleasure in work.* When management makes its particular industry a place where it is a pleasure to work, friction with labor will be considerably reduced.

8. *Maintenance of good records.* Fulton[1] states that records are the "seeing eye of industrial medicine. . . . . Of all the tools used by industry, the human tools are the most important. They cannot be standardized, maintained, or improved by any totalitarian panaceas. There is but one way to deal with them and to recognize each one as a unit of physical and spiritual personality. The first duty of medicine is to be individual. Therefore, in recording their experiences, industrial medical departments should make the INDIVIDUAL their FIRST AIM."

One of the best contributions which a large clinic can make to medical knowledge is a statistical one; from observation, tabulation, and comparison of data concerning a large group of people, one is able to deduct certain facts. Industrial medicine offers this opportunity. Again to quote Fulton: "Where is there another such clinic (than the aggregate of industrial plants) to study not only the injured and the sick, but the safe and healthy as well."

Good records also have an economic value to industry. If adequately and intelligently planned, they will easily convince management as well as labor of the value of a medical program. The type of records to be kept and their scope varies with the kind of work they are intended to record. Each medical director should design a system suitable to the requirements of the situation but always adequate to fulfill their purpose, for otherwise they are useless and the time spent in keeping them is wasted. If, for instance, records do not readily reveal the source of absenteeism, the areas of accident frequency in a plant, or information useful for the safety engineer, personnel director, or industrial relations official, they fall short of their purpose (Fig. 1).

Much too often, industrial medical records note only facts directly concerned with the specific complaint which brought the workman into the plant dispensary. Yet the physician or nurse could frequently place on record other valuable observations which, however, are extraneous to that specific visit. In illustration of this point, Fulton again offers an example. "I hold the medical department second to no other industrial department as a source of safety promoting material. An accident does not begin with the injured man. It frequently forecasts itself by nothing more than a remark. Here is an example. A workman is in the dispensary, having a minor abrasion dressed. The nurse notices his torn shoe and a friendly questioning brings forth the following exclamation, 'Boy! What that could have done to my foot!' The workman was referring to an exposed chain pulley on the floor of the plant where the cogs were not covered and where he got his foot caught but fortunately withdrew before he was hurt. This and

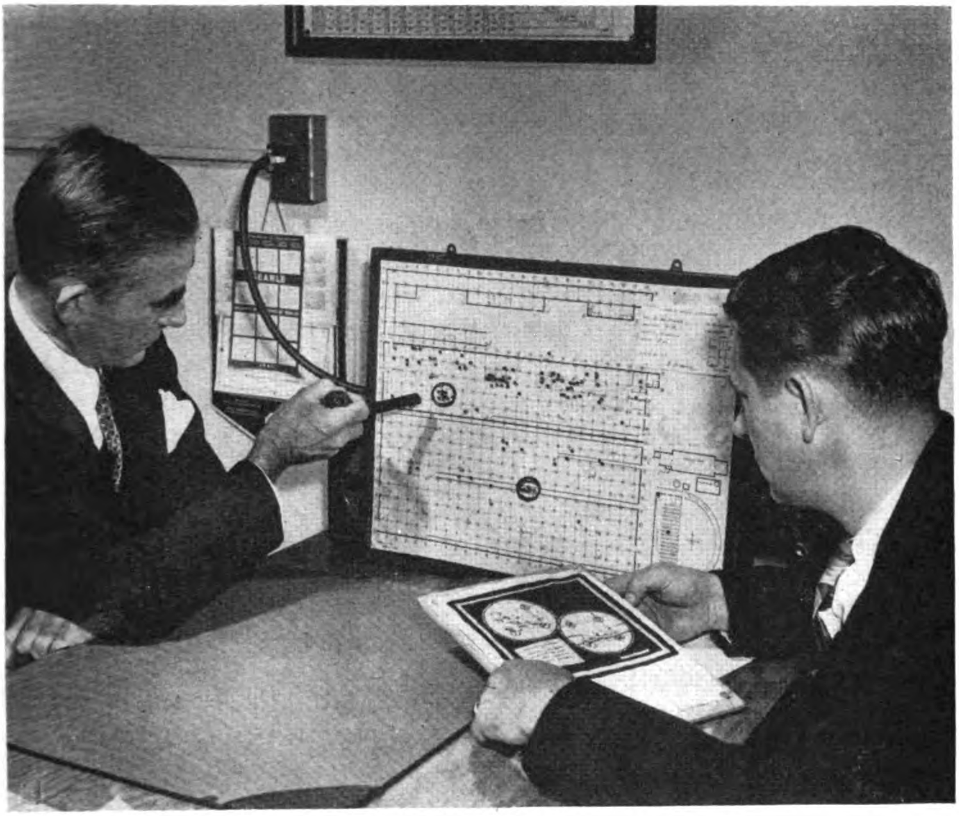

Fig. 1.—Medical and safety heads noting where frequency or repeated accidents occur in the plant. (Courtesy General Motors Corporation.)

every similar remark made in the dispensary is a signal for immediate contact with the safety engineer."

9. *Cooperation with the community physician.*

10. *Connection with approved hospitals.*

11. *Making the plant and the plant medical facilities available to medical schools for teaching purposes.*

In the preceding paragraphs the attempt has been made to describe the function of a physician and a medical department *within a plant.* But since the majority of all plants have no doctor or plant medical service, much of the care of the workman with an industrial injury or an occupational illness falls upon the physician practicing outside the walls of industry. What are his functions? How can he best fulfill his obligations to the workman, the employer, or the insurance carrier? The following points are for his consideration.

1. The Application of the Expressed Intention of Workmen's Compensation. According to the meaning of the act, all industrial cases resolve themselves into three groups:

(a) Those obviously of occupational origin (compensable)

(b) Those obviously not of occupational origin (noncompensable)

(c) Those whose occupational origin is obscure, doubtful, or not readily discernible (questionable compensability)

An example of the first group is that of direct trauma where the facts of injury are so evident as to leave no question concerning the origin of injury. Occasionally, an occupational illness will fall under the first group. Into the second group belong those which have their origin outside of the plant. But the vast majority of occupational disease claims are classified in the third group.

2. The Establishment of a Diagnosis. The first duty of a physician in handling an occupational disease claim is to make a diagnosis. He must do this in order to ascertain the liability; that is, to determine whether the employer is liable for treatment and compensation. Second, diagnosis must be established to institute proper treatment. The diagnosis must never be assumed. The basis of approach to the diagnosis of an occupational disease is discussed in the subsequent chapter.

3. The Application of a Sound Method on Reporting an Occupational Disease Claim. In cases of direct trauma, the surgeon is not called upon to delve into the origin of the injury, the nature of the offending substance, or its known effect upon the body. For that reason, the traditional and customary "Surgeon's First Report" and the "Surgeon's Final Report" are usually sufficient in traumatic cases. In contrast, no such short form is adequate in reporting an alleged or actual case of occupational disease, because such a form does not permit a detailed history or physical examination, contains no space for the

results of the environmental or laboratory investigation, and, finally, has no room for the necessary discussion of all the factors involved. All these various observations, deductions, and conclusions require a letter to the insurance carrier or to the employer, if he is self-insured. An illustrative approved form of such a letter is inserted later.

4. Adequate Case History. In the practice of no other medical specialty is the meticulous search for all available facts equally important as in a case of alleged occupational disease, and this for several reasons. In the first place, the average workman will tell the industrial physician what ails him instead of inquiring of the doctor what is wrong. In general medicine, the patient may complain of flatulence, burning, and other gastric symptoms indicating an ulcer, but he does not usually know the nature of his disease. In contrast, the average workman confronts the physician not with symptoms but with a diagnosis; that is, he will commence his interview by stating: "Doctor, I got lead poisoning."

Second, although the patient is certain as to the diagnosis, he knows but little of the details of his occupational exposure. Many are insistent that they have been handling a "poison" but cannot state its nature. To ascertain all the facts will consume much time on the part of the doctor who desires to obtain all relevant facts.

It is the opinion of the author that a good history is obtained most easily by permitting the patient to tell his story without interruption other than an occasional question to guide his account or to prevent undue verbosity. The average history obtained by writing down the patient's statements verbatim will be lacking in continuity. Furthermore, if the patient, starting to tell his story, sees the doctor vigorously writing, he may suspect that the information is "for legal purposes" and not for the sake of medical diagnosis. In that case, he is apt to "freeze" and guard every utterance, thereby depriving the physician of the full knowledge of all the facts. Following the patient's uninterrupted statements, the physician should then record the history as given and question the patient regarding any points which are not quite clear. In reporting the case to the insurance carrier or employer, it is desirable to relate the history in the words of the patient.

5. Complete Physical Examination. It almost seems impertinent to intimate that the physical examination in any occupational disease claim should be most complete. However, the fact that too frequently medical opinion, unaccompanied by either a good history or a complete physical examination, is offered in court impels the suggestion that physical examination as well as history should be most thoroughly carried out. On the very day on which these words are written, a workman was seen who honestly believed that loss of weight, constipation, blood in the stools, etc., were the result of his exposure to muriatic acid. The

physician whom he first consulted accepted the man's explanation. But simple examination of the rectum revealed a cancer. This illustration may seem absurd, but it is a typical example of the profession's indifference toward the etiology of occupational diseases.

6. The Laboratory Investigation. Very few of the occupational diseases can be diagnosed without recourse to laboratory findings. This information has its value in negative findings as well as in positive—if not more so. Some physicians fail to utilize the diagnostic possibilities of the laboratory to its fullest extent, because they assume that the insurance carrier will not pay for such procedures. This is not the fact. On the contrary, it is the thorough investigation which they most desire, for the more scientific the examination, the more clearly can their liability or absence of liability be established.

7. Estimation of Disability. It is not always realized that the insurance carrier or the self-insured employer is required by law to set up a reserve fund, sufficient to guarantee the payment of all financial liability arising from industrial injury. This reserve fund is similar to the one required of life insurance companies and is dependent upon the estimation of the aggregate cost of medical care and compensation of claims made in a given period. The financial status and security of each insurance company or self-insured employer is subject to investigation by state auditors at any unforeseen time. A reasonable degree of accuracy in the determination of the compensability of industrial injuries can only be attained if the physician's first report to the insurance company indicates the estimation of the probable temporary disability, the length of time during which medical care will be required, and the probable, possible, or certain amount of permanent disability. It is the duty of the physician to make such estimations in his first report, and to revise these from time to time in supplementary reports, if revision is indicated.

The author now proposes to illustrate the points previously discussed in this chapter by reproducing a letter from his files. It has proved good practice that all letters to the employer or to the insurance company should be written in triplicate. At the conclusion of the following letter, its more important features will be shortly analyzed.

2-16-46

Pacific Employers Insurance Company,
1033 South Hope Street,
Los Angeles 15, California.

Re: George Lopez, Employee,
M. P. Kay & Sons, Employer,
Injured (onset) 1-17-46.
Control No. 224-B.

Gentlemen:

The above captioned individual first reported to this office on 2-5-46. He is a 59 year old Mexican male, who gives his address as 2216 Pallo Ave., Monterey Park, California, and states that he is an employee of the M. P. Kay Company.

*History.*

The patient speaks fairly good English and was readily understood. Portions of his history were augmented by his daughter who accompanied him to this office. Mr. Lopez states: "The doc at the plant says I got lead poisoning. Along about the first of January—maybe before that a little—I noticed my left hand felt funny. It seemed dead—I couldn't grip things with it. One morning I noticed I couldn't even lift my coffee cup. My hand didn't want to seem to lift up—see, like this—I can't raise it. Then my right hand got the same way. I didn't know why. It kept getting worse so I went to the boss—I tell him and he tell me to see the company doctor. The next day I went to the doc and he examine me and then take some blood from my finger. A couple of days later he tell me to come see you. It is funny—I feel good except for this." At this point in the history, the daughter stated that her father had complained in the past week or two that he had a weak feeling in his legs.

*Referable Symptoms:*

Head and Neck: Patient denies any headaches, dizziness or tinnitus. He had for many years a periodic discharge from the left ear. States he can read and see well without glasses. Has had no dental care for many years. Denies any throat trouble.

Respiratory: Denies cough, pains in chest or frequent respiratory infections.

Cardiac: No dyspnea, orthopnea, palpitation or cardiac pain.

Gastro-Intestinal: Denies flatulence, colic, burning sensations, constipation or diarrhea.

Genito-Urinary: Nocturia 2 or sometimes 3 times; no frequency or dysuria. Denies venereal disease.

Nervous System: Sleeps well; is not nervous or irritable.

Weight: Stationary for years, until the past several months. Thinks he has lost about 12 pounds.

*Personal History:* Patient is a widower who lives with his daughter. Wife died 5 years previously following an abdominal operation. Denies any family history of tuberculosis, cancer, diabetes or insanity. Drinks wine; no whiskey or beer. Does not smoke.

*Past Medical History:* Patient states that with the exception of an occasional cold he has never been sick. Daughter testifies she cannot remember her father being sick.

*Occupational History:* From very early manhood the patient was a rancher in Ventura County. He continued in this type of work until 1941, when he took a job as stevedore at the Naval Base near Ventura, California, where he worked until August of 1945. In that month he went to work for the M. P. Kay & Sons Company. His work for this company consisted of wheeling sealed drums of "lead dust" from the store house to the loading platform. He has engaged in no other type of work for this company.

*Present Complaints:*

1. "My hands feel weak. I have no grip in them and I can't lift them."
2. "For the past several days I have noticed my legs are weak—maybe longer than that."
3. "I tire out more than I used to do."

*Physical Examination:*

The patient is a small, undernourished, 59 year old Mexican male whose weight is 115 pounds, and height five feet three inches. His skin is of a pale yellow, sallow color. He does not appear to be in any discomfort.

Head: The size and shape are normal. The pupils are round, equal, and react to light. Funduscopic examination normal. The left ear canal contains white desquamated material which occludes the drum. The right ear canal and drum are normal. Nose and throat negative. Teeth: many are missing, while the remaining teeth reveal cavities in the upper right and lower left first molars. Moderate pyorrhea with a fine bluish stippling along the anterior gingival margin.

Chest: Small, rachitic in appearance with emaciation of the interspaces. There is no lag of either side on deep inspiration. Percussion note resonant throughout; on auscultation no rales or friction rubs are heard. Breath sounds normal.

Heart: Rate of 88, regular, no murmur heard, no apparent enlargement. Blood pressure 140/86.

Abdomen: Flat, thin muscles. No organs or masses palpable; no areas of tenderness or rigidity. No hernia.

Genitalia: Normal.

Prostate: Slight bilateral enlargement.

Extremities: On inspection the bilateral wrist drop is apparent. When he extends his forearms all attempts to raise his hands reveal no visible movement. There is considerable bilateral atrophy of the extensor group of muscles. No atrophy of either upper arm; nor is there any atrophy of the lower legs. Motions of the ankles are normal. The gait is normal.

Neurological Examination: Entirely normal except for the involved forearms. Here the extensor reflexes are absent. The tactile and thermal sensibility is also diminished. There is no sensory disturbance of the lower legs despite the patient's claim of weakness.

*Laboratory:* Blood Count:

| | |
|---|---|
| Hb. | 63% or 10.7 Gm. |
| Color index | 1.0 |
| Erythrocytes | 3.160 000 |
| Leukocytes | 7.600 |
| Polynuclears | 64 |
| Lymphocytes | 34 |
| Monocytes | 2. |
| Stippled erythrocytes/million | 16.000 |
| Blood lead determination | 0.41 mg./100 cc. of bl. |
| Urine lead determination | 0.39 mg./liter |
| Blood Wassermann | negative |

Urine—1018, clear, amber, albumen: trace, sugar: negative, microscopic: negative.

*Discussion:*

The fact that this patient relates an insignificant exposure while working for your insured company, demanded a more exhaustive study than that indicated in the above report. He was therefore kept in the hospital for two weeks while the above tests were repeated under meticulous management. The blood and urine lead determinations were repeated three times, and all results were comparable to the first, although insignificant drops in the lead content in the blood and urine occurred.

This is only the second case of wrist drop due to lead absorption that the writer has seen in the last ten years. This phenomenon is rare compared to the frequency of other signs and symptoms of lead intoxication. When present, it is usually after prolonged and severe exposure to lead, and is believed to be a late manifestation of this illness. It is usually preceded by other manifestations which serve as a signal for the workman to discontinue his exposure.

Also, in view of the patient's statement that he did not work where there was any exposure to lead, but that he merely handled sealed cans of "lead dust", I inspected the plant where he has been employed. I was conducted on my tour by the personnel manager who took me to a large store room where these cans were stacked and from where the patient would truck them to the loading platform. Although there was an occasional thin, almost imperceptible layer of dust on some of the cans, it did not appear that the patient could have had any real exposure on this job. The personnel manager assured me the workman had performed no other work at the plant.

Still at a loss to account for this apparent excessive absorption, I went to the home where he lived to inspect the plumbing. The home was of modern construction, the pipes which conducted the water were traced to the local distributor and found to be free from lead. The daughter informed me that her father drank a great deal of wine, but that he did not make it himself and that he bought various kinds and from various wineries.

Still not satisfied, I returned to the plant but on this second visit I went about unaccompanied by anyone from the front office. I talked with different employees who knew Mr. Lopez, and discovered some pertinent facts. It appeared that Mr. Lopez was not rushed on his job and that it afforded him frequent periods for rest. During these periods, which were three or four times daily, he would wander into the smelter room where he had several Mexican friends working. One of these tapped off the slag. The room was exceedingly dusty and all men who worked there wore masks. He also was prone to visit another friend of his who was running the lead dust into the containers before sealing them. Here again, the workers in that department were protected, but Mr. Lopez was not.

With the new facts ascertained, air analyses of the several spots which Mr. Lopez visited were made. It was found that at the packing area the air sample contained 2.1 mg. of lead per 10 cubic meters of air; the area of tapping off contained 2.0 mg. of lead per 10 cubic meters of air. (The accepted threshold is 1.5 mg. per 10 cubic meters of air).

*Conclusion:*

As the insurance carrier for the M. P. Kay & Sons Company, you are hereby informed that their employee, Mr. George Lopez has lead intoxication which is of occupational origin. He has been placed under treatment. In view of the type of disability it is doubtful whether the ordinary type of treatment will reduce the existing disability to any degree.

*Disability:*

The patient has a total temporary disability. It is likely that he will retain a total permanent disability.

You will be informed every two weeks by supplementary report as to his condition.

Yours very truly,
R. T. Johnstone, M.D.

The foregoing letter was chosen because it illustrates many excellent teaching points. The case falls under the classification in which at first the occupational origin of injury was not readily discernible. Despite the evidence of wrist drop and the laboratory evidence of abnormal lead absorption, the source of the lead had to be determined. This required persistence in running down facts in the occupational exposure, which at first were not evident. For reasons of space in this chapter, successive laboratory reports were deleted. It will be noted that not only laboratory studies of the patient were made, but that also environmental studies were conducted. These proved important not only in the determination of the occupational origin of the intoxication, but also induced the employer to correct his plant hygiene and protective measure.

The first portion of this letter exemplifies that the occupational disease history and physical examination must be as complete as in any other type of clinical medicine. But an added feature for a letter of this type not commonly found in the ordinary medical report is the part allocated to discussion. It is in the discussion that findings are freely evaluated, peculiarities of the case are discussed, and an attempt is made to show why the case is or is not of occupational origin; in other words, why it is compensable or noncompensable. Comparable to the rebuttal period following a debate, the pros and cons must be evaluated in terms readily understandable to the insurance carrier or the Industrial Accident Commission in the eventuality that the case should become the subject of a medicolegal controversy.

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# CHAPTER V

# THE TEACHING OF INDUSTRIAL MEDICINE

If education is to fulfill the requirements resulting from a changing social and economic philosophy, it must be in harmony with the constantly shifting pattern of *vocational needs.* While the biologic inheritance of a race remains practically unchanged from age to age, education is always related to some concrete and evolving social situation. Hence the problem of education assumed one form in ancient Athens, yet quite another one in twentieth-century America. What was adequate in our father's time has now become inadequate. It is clear, therefore, that any group charged with the task of shaping the educational theory or practice for any people should consider the trends and interests of the society it serves. Medical education is no exception to this truth.

To the credit of medical education it can be said that it has kept pace with scientific progress and has well prepared its students for the practice of general medicine. But it has been extremely dilatory in recognizing the specific requirements inherent in industrial health. In fact, for all practical purposes, it has ignored the urgency for this type of teaching. One objection which has been raised against the inclusion of industrial health in the medical school curriculum has been that the curriculum is already overcrowded—which is a valid reason. But education of any type, medical or otherwise, must constantly eradicate antiquated subjects to make room for that which is currently important. And the fact that industrial medicine is now a subject of major importance cannot be denied.

As a member of the Committee on Education of the American Association of Industrial Physicians and Surgeons, as well as a member of the Committee on Certification, the writer has discussed the subject of teaching industrial medicine with many deans and professors of our medical schools. While some of these are in accord with the ideas expressed in these pages, it is amazing to see how many still insist that industrial medicine is no different from the practice of general medicine, except that it is practiced in the plant instead of in the home or office. Such a viewpoint envisions only the treatment of the industrially ill or injured workers. It does not admit the fact that industrial medicine deals with groups as well as individuals and that its chief concern is prevention rather than treatment.

Where is the physician in private practice who is concerned with lighting, noise, vision, ventilation, fatigue, nutrition, humidity, temperatures, radiant energy, or the toxicity of chemicals. With exception of the field of childhood immunization, or the periodic gynecologic examination of female adults, probably not 5 per cent of general medical practice is preventive. In strict contrast to this, 70 per cent of industrial medicine is preventive. Industrial medicine is dealing with society in a very concrete manner. This alone is sufficient reason why the teaching of industrial medicine is an urgent necessity in the curriculum of every medical school.

Disrupted by the accelerated program compelled by the war, and now in the throes of stabilizing its curriculum, it cannot be expected that any medical school will be able to create immediately an adequate department of industrial health. But until the time arrives when industrial health will be accorded its rightful place in the curriculum, the teaching of that subject should be made a component of each clinical course. In the first issue ever devoted to industrial medicine by the *Journal of the American Medical Association,* the present writer[1] described the manner in which this could be done. It was suggested that a physician rich in industrial experience should be made director of the department and accorded the position of a full clinical professor; that he should endeavor to gain the cooperation of the many plant physicians in the community who could provide him with teaching material; that he then assign this material to the various departments. In this manner, lead intoxication could be studied by classes in hematology and intestinal diseases; industrial skin lesions would be assigned to the department of dermatology; the workman with a cough, loss of weight, hemoptysis, etc., who worked in the dusty trades, could be studied jointly in the department of pulmonary diseases and roentgenology. Thus a systematic program of schooling medical students in all the varied phases of industrial medicine could be started.

Obviously, the aforementioned plan is only a first step in the right direction. It is still woefully incomplete and lacks the inclusion of laboratory investigation, the study of plant populations, working environment, or research.

Those charged with the planning of medical school programs will have to develop a course of instruction in industrial medicine along the following lines suggested by Kehoe.[2] "Constant research is the key to the practice of industrial medicine and the role of investigation is much more vital than has been suggested. Not all investigation is to be carried out in the laboratory. Indeed, these must be a continual correlation between laboratory studies and systematic clinical observation, if either is to be effective. For example, the limits of safety with respect to the exposure to the vapor of an industrial solvent may be determined tentatively by laboratory experiments carried out upon animals. This should be a general practice. But it is also necessary to establish the facts with reference to human exposure to such materials by human experience under carefully controlled conditions." "As industrial production is continually changing, so the industrial environment and its hygienic problems are shifting. For this reason, investigation must be extensive and continuous, if it is to keep up with current developments. Obviously, therefore, the factual basis of medical practice must be dependent upon the productivity of research."

In a personal letter from Kehoe[8] he voiced agreement with the writer as to the necessity of collaboration with the colleagues teaching clinical medicine and stressed that such a combination will prove of great benefit to a teaching program connected with a research institute "with the best facilities obtainable in apparatus and personnel in the fields of theoretical and applied science; . . . . of continuing intramural physical, chemical and physiological investigations."

To those interested in medical education it should be apparent from the discussion in this chapter that no course in industrial medicine can have the desired effect if it is purely didactic. Students should be allowed to acquaint themselves with clinical cases for the purpose of study and should subject such cases to differential diagnosis. They should devote some hours to plant surveys and be afforded an opportunity to observe industrial processes. A desirable inclusion in any program would be to assign a student in his senior year to an internship of at least two weeks' duration in an industrial plant medical department.

During the past few years, outlines for a course in industrial medicine have been formulated by sundry individuals or groups. It is a grievous error, however, that almost without exception these plans fail to make any provision for the teaching of laboratory procedures in industrial toxicology and their interpretation. But unless this subject matter is included, no course can be considered as basic or complete.

The outline which follows is one prepared by the Committee on Medical Education of the American Association of Industrial Physicians and Surgeons. It is offered here not as a perfect system to be adopted by those newly assigned to this field, but rather in the hope that many original ideas may be added to it by these teachers.

*Content and Organization of Teaching.*—Experienced physicians in industry have testified that industrial health activity falls into three principal categories:

1. Industrial medical administration
2. Industrial hygiene and toxicology
3. Industrial medicine and traumatic surgery

I. **Industrial health administration**
  1. Origin and later development of industrial health
    (a) Clinical interest in occupation and health
    (b) Legislative background

        - (1) Factory acts
        - (2) Workmen's compensation
        - (3) Social security

2. Occupational morbidity and mortality
    - (a) Causes and duration of lost time in industry
        - (1) General experience
        - (2) Experience in specific industries
    - (b) Reportability of industrial accidents and diseases
3. Distribution of industrial health service
    - (a) Scope and objectives of industrial health
    - (b) Population groups involved
        - (1) According to plant size
        - (2) According to type of industry
    - (c) Costs of medical service in industry
    - (d) Economic and other benefits
4. Classification of industrial physicians
    - (a) On-call and part-time (detached)
    - (b) Full-time (attached)
    - (c) Consulting services
        - (1) Industrial hygiene
        - (2) Clinical specialties
    - (d) Qualifications for industrial medical service
5. The industrial medical department
    - (a) Organization
        - (1) Personnel
        - (2) Plant and professional relations
    - (b) Quarters and equipment
    - (c) Specific functions
        - (1) Treatment and hospitalization
        - (2) Prevention and sanitation
        - (3) Health conservation
            - (a) Physical examinations
            - (b) Health education
        - (4) Rehabilitation
    - (d) Special medical problems of small plants

## II. Industrial hygiene and toxicology

*Course Content*

1. Industrial health exposures
    - (a) Classification
    - (b) Essential toxicology
    - (c) Safe concentration codes

2. The plant survey
    - (a) Identification of exposures
        - (1) Medical procedure
        - (2) Engineering procedure
    - (b) Control of exposures
        - (1) Medical
        - (2) Engineering
    - (c) Maintenance of control
    - (d) Records
3. Plant sanitation and hygiene
    - (a) Water supply
    - (b) Waste disposal
    - (c) General housekeeping
    - (d) Illumination
    - (e) Ventilation and air conditioning
    - (f) Noise and vibration control
4. Personal hygiene for workers
    - (a) Toilets and washrooms
    - (b) Fatigue control
    - (c) Nutrition
    - (d) Housing
    - (e) Recreation
5. Coordination of industrial and community health service
    - (a) Communicable disease control
    - (b) Industrial waste control
    - (c) Vital statistics
    - (d) Safety codes and regulations
    - (e) Factory inspection and regulation

III. **Industrial medicine and traumatic surgery**

*Course Content*

1. The worker and the job
    - (a) The worker
        - (1) Physical and mental fitness
        - (2) Aptitude tests
        - (3) Medicine and personnel relations
    - (b) The job
        - (1) Physical and mental requirements
        - (2) Duration—hours and wages, day, night, peak loads
        - (3) Special processes, equipment, and working conditions
        - (4) Environmental factors

2. Industrial accidents
    - (a) Incidence and costs
    - (b) Classified causes
    - (c) Emergency care and transportation
    - (d) Surgical management
    - (e) Prevention
    - (f) Estimation of disability
    - (g) Records
3. Occupational diseases
    - (a) Etiologic classification
    - (b) Pathology and toxicology
    - (c) Medical management
    - (d) Prevention
    - (e) Disability evaluation
    - (f) Records
4. Nonoccupational disability
    - (a) Incidence and costs
    - (b) Classified causes
    - (c) Medical advisory service
    - (d) Follow-up service
    - (e) Records
5. Workmen's compensation and rehabilitation
    - (a) Administrative methods
    - (b) Medical relations and regulation
    - (c) Insurance practice

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2. Kehoe, R. A.: Medical Education, Research and Practice in Relation to Industrial Health, Med. Press & Cir. **215**: 8, 1946.
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# CHAPTER VI

# THE BASIC APPROACH TO A DIAGNOSIS OF OCCUPATIONAL DISEASE

"Lack of systematic personal training in the methods of recognition of disease is apt to place us on the level of empirics and quacks" wrote the Dean of American medicine at a period when medicine was more art than science. Since Osler's day, clinical teaching and scientific implements have elevated diagnosis in general medicine to a level of undeniable proficiency. But this same degree of proficiency is not attained nor even frequently attempted by the general practitioner or specialist when he finds himself confronted with an actual or alleged occupational illness. In no other phase of medicine will empiricism be found to be so prevalent.

It is not difficult to understand why this incompetence exists with regard to the diagnosing of occupational diseases. One reason which has been previously emphasized is the neglect of this subject in undergraduate and postgraduate teaching. But this is far from being the only excuse. The chief reason is the marked indifference which the average doctor has often manifested concerning the "diseases of workmen." This attitude undoubtedly stems from experience in the past when only on rare occasions a patient with an occupational disease would cross the average physician's doorstep. But times have changed! The industrialization of every section of the United States and the acceptance of the responsibility for industrial health by management makes it inevitable and inescapable that the entire medical profession should acquaint itself with occupational medicine.

The initial approach to the diagnosis of an occupational disease is not dissimilar from that used to diagnose any disease of communal origin, since the noxious industrial substances are capable of affecting any of the internal organs or systems, according to their specific action. In the initial approach to a new case, the history is the first important step. This history must be meticulous as to detail, and all-inclusive. It should contain every occupational activity since the patient's school days. Unless such a comprehensive history is obtained, erroneous diagnostic assumptions will be almost unavoidable.

To illustrate, from a patient whose signs and symptoms suggested bronchial asthma, a physician obtained a history stating that he was currently working as a baker and had been so employed for six years. Prior to that he had been an apartment house manager. His x-ray films revealed a moderately diffuse fibrosis of the lungs with evenly scattered nodulation. In reporting the case to the employer, the physician correctly attributed the asthma to a sensitivity to flour dust, but he incorrectly stated that the fibrosis noted on the x-ray films was due to the inhalation of this flour dust. In his conclusions the physician committed two errors: (1) flour dust produces no perceptible, if any, fibrosis of pulmonary tissue and (2) the occupational history was inadequate. Had the physician inquired further into his patient's occupational history, he would have found out that the man had been a hard rock miner for twenty years, a type of work which had produced this fibrosis.

Then there is the case of the workman whose marital status was rendered temporarily stormy when, during his examination, it was noted that he had a perforated nasal septum. In the presence of his wife, this patient was advised to have a Wassermann test made and it was intimated that the perforation indicated the possibility of syphilis. Actually this perforation had been caused when the patient, as an electroplater, had been exposed to chromic acid, a fairly common cause of nasal perforation.

Nor should a workman's statement of his occupation be accepted as descriptive of his industrial exposure. It is most important that the physician inquire carefully into all the various procedures and substances pertaining to the stated occupation. Men are prone to classify themselves by a title which does not reveal the actual nature of their work. Indeed, workmen are rarely aware of the materials utilized in the processes in which they are engaged.

An electroplater, for instance, may be exposed to one or more of some twenty chemicals such as arsenic, antimony, benzine, benzol, cadmium, carbon disulfide, carbon tetrachloride, chromium, cyanogen compounds, hydrofluoric acid, sulfuric acid, lead, mercury, etc. Operating a turret lathe is not always a simple mechanical process, for often, in order to obtain a smooth finish, carbon tetrachloride may be squirted onto a hot surface. A welder may be exposed to lead or a combination of metallic oxides, or to nitrogen dioxide or carbon monoxide.

To assume that a workman has an occupational illness is empiricism; to prove it is a scientific procedure. To do so often requires as much leg work as brain work. The physician who wishes to learn the cause or source of an occupational disease must often leave his comfortable office, cross the tracks or go across town to the plant to investigate the working environment of his patient. In general medicine the hospital ward is the classroom out of which comes much of our clinical knowledge; in industrial medicine it *should be* the plant.

The following is an example proving how easy it is to be misinformed. The author, in attempting to determine the cause of a severe anemia in a patient, telephoned the owner of a small wartime industry, only to be assured that the process in which the patient was engaged used no benzol but rather a petroleum distillate. Not satisfied as to the accuracy of this information, a trip to the plant disclosed that the suspected solution bore a trade number but no label indicating the nature of its ingredients. It was necessary to write to the manufacturer, from whom it was learned that the solvent contained 40 per cent benzol. In this instance, the employer had not been aware that his superintendent had made a substitution several months previously.

Likewise, the failure to obtain accurate knowledge regarding the nature of a workman's exposure will often render treatment useless or even harmful. A physician practicing in the neighborhood of a company manufacturing plastics was at loss to explain why some mild cases of dermatitis became aggravated following the application of calamine lotion, and why further aggravation occurred when he switched to a "soothing ointment" containing menthol. Had he taken the time and trouble to inquire at the plant, he would have learned that the offending substance in the plastic process, which had caused the original dermatitis, was either phenol or formaldehyde or a combination of these, both of which are carbon derivatives. Knowing this, he would not have prescribed medication also containing derivatives of a similar nature.

Having secured a complete occupational history, and having determined beyond any reasonable doubt *the exact substance* to which the workman was exposed, and which is now suspected to be the offending agent, the next step must be to subject this suspicion to the following inquiry:

1. Is the suspected substance known to have noxious properties? Or is the suspected substance known to be inert or relatively so? A great many substances normally handled in industry are harmless. This applies to many metals, alloys, and dusts. Most metals or alloys like aluminum, cadmium, magnesium, and others cause no harm when they are in the so-called "cold state," such as sheet metal, bearings, and the like. But certain metals, such as cadmium, when subjected to hot temperatures, liberate oxides which may not only be toxic but lethal. The toxicity of solvents is also increased by heating. With few exceptions the industrial dusts such as textile dust, or dust from flour, wood, feathers, etc., are harmless, since they either fail to pass through the protective mechanism of the upper respiratory tract or, if they do so, contain no property capable of producing pulmonary reaction. It is well known that men exposed to cement dust over a period of twenty or thirty years reveal only minimal, nondisabling fibrosis. Yet, under similar working conditions, a dust containing free silica could not be expected to have such a negligible reaction. The toxicity of solvents also varies greatly; the toxic potentialities of gasoline cannot be compared to benzol. In other words, the physician must be acquainted with the toxic or nontoxic qualities of any given industrial substance under consideration.

2. What is the degree or concentration of the exposure? As a corollary of the previous paragraph, it must be understood that the mere presence of a so-called noxious substance does not necessarily mean that an exposed individual will be affected by it. The human body is capable of detoxifying poisonous substances, and because of this ability a level exists at or below which absorbed substances are tolerated by the body without any harmful consequences. We know that this occurs in the detoxifying of the toxic products of normal metabolism. With this ability of the body to sustain without harm the intake of certain chemicals or noxious materials, hygienists and toxicologists have set up a standard of permissible limits of safety for all, or almost all, of the solvents, gases, vapors, or dusts in industry. By animal experimentation and clinical observation, information has become available to facilitate the safe handling of these substances. Therefore, in the diagnostic approach, it behooves the physician to ascertain in any given instance the circumstances of the alleged exposure, to determine whether the specific substance was present in amounts within the accepted limits of safety, or above these normal limits.

3. To what degree can a noxious substance be absorbed? How rapidly can it be eliminated? Although a few industrial materials may be absorbed through the skin or by ingestion, the vast majority enter the body by inhalation. With regard to the absorption of volatile substances through the lungs, Henderson and Haggard[1] state: (1) "No more of the substance can be absorbed than is taken into the lungs in the air breathed and (2) absorption ceases when an equilibrium is reached between the amounts in the body and in the air." These two important facts are often overlooked when an estimation is made of the degree to which exposure to the gases has affected the patient. A more detailed discussion of this subject and of the action of gases will be contained in the introduction to Part II.

4. Following an undue exposure to a noxious substance, how soon should evidence of an illness be expected? With few exceptions, intoxication from an industrial chemical will become noticeable during the period of exposure. Because of the law of elimination, a delayed reaction does not usually occur after the patient has been withdrawn from exposure. Silicosis or radium poisoning, however, are notable exceptions. Some reports in the literature even suggest the possibility that benzol may produce an aplastic anemia, leukemia, or a neoplasm several months or years after exposure, but more abundant and reliable confirmation of this point is still required.

5. What organ or system of the body is most likely to be affected by an undue exposure to a given industrial material? In general medicine, infection, organic dysfunction, or altered metabolic states produce a fairly constant clinical pattern established by experience,

physical examination, laboratory findings, and pathological studies. A similar foundation of facts exists in industrial medicine. Experience has taught us that from an undue exposure to a given substance we can expect a typical reaction. Any variation from the usual picture should induce doubt regarding the occupational origin of the illness. Adherence to this guiding principle will prevent unfounded speculation and conjecture.

To emphasize the aforementioned point, one could classify the noxious substances in industry according to the systems of the body which they affect, such as those which attack the respiratory tract, or the central nervous system, or the blood-forming organs, or such organs as the liver or the kidneys. Such a classification should differentiate between the acute and the chronic intoxication. Benzol, for instance, affects the central nervous system if the exposure is sudden, short, and severe; but if the exposure is not acute but chronic, then the central nervous system escapes and the effect is upon the blood-forming organs.

Obviously, then, this predilection of a given chemical for a certain organ or system suggests where the major emphasis should be placed in the physical examination and laboratory investigation of the patient. A Negro who had been dipping metal parts into a tank of trichlorethylene for eight hours without adequate protection went to a physician at the end of his shift for chemical burns of the hands. While applying an ointment to the hands, the doctor noticed that the patient seemed groggy and somewhat incoherent. He made a note on his record that the patient appeared "drunk." The patient was sent home following the treatment of the burned hands, but one block away from the doctor's office he collapsed and was picked up by a policeman and sent to the county jail for drunkenness. He was intoxicated but not from alcohol; it was from trichlorethylene. Although the physician was apprised of the substance to which the workman had been exposed, he did not take into consideration that the central nervous system could be affected by this solvent. The patient should have been given a complete physical examination with special attention to the nervous system.

6. The laboratory investigation. Likewise, an appreciation of the type of reaction likely to follow an intoxication from any given substance will guide the nature of the laboratory investigation. Experience has taught that certain noxious agents have a selective action upon certain systems or organs so that, with this selective action in mind, the laboratory investigation likewise becomes selective instead of at random. It is also necessary to remember that with certain poisonings, no type of laboratory confirmation can be considered specific. The time interval between the last exposure and the time of examination will equally have to be evaluated.

In the following chapter, an outline will be presented to aid the reader in choosing the right laboratory tests for the determination of certain suspected intoxications, or to obtain some confirmatory aid.

7. Plant investigation. In a systematic approach to the diagnosis of occupational diseases, plant inspection must be included. It is not expected of the average physician that he should have training in industrial hygiene or engineering. Nor is it expected of him to be able to use the various implements employed to collect dust or measure the concentration of vapors or gases. For such specific determinations he can take recourse to the Industrial Hygiene Division of the United States Public Health Service, or to a number of commercial organizations capable of making industrial hygiene surveys. But the average physician can learn the primary principles of industrial hygiene. This self-education combines instructive reading with frequent surveys of those plants to which the doctor may have access. Such surveys teach the value of local exhaust systems, of personal protective equipment, of general plant ventilation, of the need for "isolating" certain processes from workers who are unprotected, and of the general efficiency or inefficiency of the entire plant hygiene as it pertains to showers, individual lockers, washrooms, individual towels, drinking cups, cluttered or unobstructed aisleways, etc.

To summarize, the diagnosis of an occupational disease should proceed step by step along well-defined methods. If such a procedure is followed, instead of the too frequently used "conjectural approach," the entities of these diseases will be established and medicolegal testimony will adhere to scientific standards.

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# CHAPTER VII

## LABORATORY PROCEDURES OF VALUE IN THE DIAGNOSIS OF THE OCCUPATIONAL DISEASES

Although some of the material to be found in the following outline will also appear in subsequent chapters, it was felt that a chart such as the one contained in this chapter would serve the reader as a quick, ready reference. As a preface to this chart, the reader is asked to recognize some of the limitations of laboratory investigation of the occupational diseases.

1. Very few tests can be made in the physician's office.

2. The facility with which it is possible to obtain quick, reliable laboratory information in general medicine does not exist in occupational medicine. Comparatively few of our hospital or clinical laboratories are sufficiently equipped or properly staffed to conduct investigations in the field of industrial toxicology, although many do not admit this incompetence. It behooves the physician to recognize this fact and to choose a laboratory of proved competence.

3. In some instances, diagnostic confirmation should not be left to a single test but rather to a combination of tests. For example, in the attempt to establish a diagnosis of lead intoxication, the complete blood count, the stippled or basophilic aggregation count, and estimations of the lead in the urine and blood should all be utilized. To determine the degree of benzol absorption, the determination of urinary sulfates combined with blood studies give more complete information than either of these tests alone.

The mere presence of a metal in the biologic tissue or fluid does not necessarily indicate poisoning. Some of these are normal constituents of the body, and for this reason it is necessary to distinguish between normal content and abnormal absorption. One must also take cognizance of the possibility of a nonoccupational exposure.

5. The fact that many volatile substances are rapidly eliminated from the body limits the probability of their detection by laboratory tests. The interval of time between exposure and examination must always be considered.

6. The physician should learn the accepted standards of a possible normal presence of these substances to facilitate his own interpretation of laboratory reports. Among the various laboratories, especially those of meager experience in this field, there is a lamentable lack of uniformity in stipulating the limits of normal.

TABLE I. TESTS TO DETERMINE THE PRESENCE OF TOXIC AGENTS IN BIOLOGIC MATERIALS

| TOXIC AGENT | BIOLOGIC MATERIAL USED FOR ANALYSIS | MEAN VALUES AND RANGE OF NORMAL SUBJECTS | METHODS OF ANALYSIS |
|---|---|---|---|
| Lead | Blood<br>Urine<br>Feces | 0.03 mg./100 ml. (0.01-0.05)<br>0.03 mg./1 (0.01-0.08) | (1) Dithizone<br>(2) Lead chromate<br>(3) Spectrographic |
| Mercury | Urine | None | (1) Dithizone<br>(2) Spectrographic |
| Arsenic | Hair (Pubic)<br>Urine<br>Feces | 30 p.p.m.<br>0-0.85 mg./1 | (1) Gutzeit mercuric bromide method |
| Manganese | Blood<br>Urine | 0.014 mg./100 cc. | Colorimetric method |
| Fluoride | Blood<br>Urine | 0.01 mg./100 cc.<br>0.2-0.4 mg./1 | Volumetric method |
| Radium | Exhaled air | None | Ionization chamber |
| Selenium | Urine | 0.04-0.05 mg./1 range (0-0.15 mg./1) | Electrotritrimetric modification of the iodine-thiosulfate method |
| Carbon monoxide | Blood | 1.5% CO-Hb (Nonsmokers)<br>3-10% CO-Hb (Smokers) | (1) Spectrophotometric<br>(2) Van Slyke-manometric method (Roughton)<br>(3) Pyrogallic tannic acid method |

TABLE II. TESTS TO DETERMINE THE PRESENCE OF CONJUGATED PRODUCTS

| METABOLIC OR CONJUGATED PRODUCT (URINE) | TOXIC AGENTS WHICH PRODUCE METABOLIC OR CONJUGATED PRODUCT | NORMAL RANGE | METHODS OF ANALYSIS |
|---|---|---|---|
| Urinary sulfates | Benzene<br>Aniline<br>Phenol<br>Xylidine | *Inorganic sulfate*<br>Total sulfate ratio, 90% | Folin method |
| Hippuric acid | (1) Toluene<br>(2) Ethyl benzene and higher homologues which are oxidized to benzoic acid | 0.6-0.7 Gm./24 hr. | Crystallize and determine amount by titration with NaCH |
| Glucuronates | (1) Phenol<br>(2) Terpenes<br>(3) Benzene<br>(4) Xylidine<br>(5) Aniline | 0.7-1.4 Gm./24 hr. | Naphthoresorcinol reaction photoelectric absorptiometer |
| Formic acid | Methyl alcohol | 30-120 mg./24 hr.<br>Average, 60 mg. | Dakin method |
| Thiocyanate | Nitriles | *Serum*<br>Nonsmokers, 0.4-0.6 mg./100 ml.<br>Smokers, 1.5-2.0 mg./100 ml.<br>*Urine*<br>Nonsmokers, 0-085 m./24 hr.<br>Smokers, 14 mg./24 hr. | Colorimetric method for ferric cyanide |
| 2, 6 dinitro–4–aminotoluene | TNT | | Method of Snyder and von Oettingen |
| p–aminophenol | Aniline | | Aminophenol reaction |

TABLE III. TESTS TO DETERMINE PHYSIOLOGIC OR ORGANIC CHANGES

| ORGAN OR TISSUES CONCERNED | AGENTS INVOLVED | NATURE OF ALTERATION | METHOD OF DETECTION |
|---|---|---|---|
| Blood-formed elements | Benzol<br>TNT<br>Arsenic<br>Sulfonamides | —a—<br>Toxic inhibition of blood-forming organs including aplastic anemia and granulocytosis | (a) Complete blood<br>(b) Reticulocyte counts<br>(c) Platelet counts<br>(d) Sternal marrow counts<br>(e) Hematocrit |
| | Aniline<br>Arsine<br>Lead<br>Nitrobenzene<br>Phenylhydrazine<br>Stibine<br>TNT | —b—<br>Hemolytic | (a) R.B.C. count<br>(b) Hematocrit<br>(c) Hemoglobin<br>(d) Plasma hemoglobin<br>(e) Fecal urobilin<br>(f) Icterus index<br>(g) Stain urinary sediment for hemosiderin in cells |
| | Radium<br>X-ray | —c—<br>Physical injury<br>(1) External irradiation<br>(2) Internal irradiation | Complete blood count<br>Bone marrow studies |
| Blood pigments | Aniline<br>Nitriles<br>Nitrobenzene<br>Phenylhydrazine<br>Toluidine<br>Xylidine | Methemoglobin (ferrous Fe in Hb oxidized to ferric Fe) | (a) Gasometric (Van Slyke)<br>(b) Spectrographic<br>(c) Spectrophotometric colorimeter |

| | | | |
|---|---|---|---|
| Liver | (1) Chlorinated hydrocarbons, such as (a) carbon tetrachloride, (b) tetrachlorethane, (c) chloroprene, (d) chlorinated diphenyls, (e) chlorinated naphthalenes<br>(2) Dinitrophenol<br>(3) Dioxan<br>(4) Trinitrotoluene<br>(5) Xylidine | (1) Fatty degeneration atrophy of liver, toxic hepatitis, or cirrhosis | Combination of liver function tests such as:<br>(1) Icterus index<br>(2) Urine bilirubin (Harrison's spot test)<br>(3) Urobilinogen (Watson's method)<br>(4) Hippuric acid excretion<br>(5) Bromsulphalein excretion (5 mg./kg.)<br>(6) Cephalin flocculation<br>(7) Alkaline phosphatase<br>(8) Thymol-barbital |
| Kidney | Arsine<br>Carbon tetrachloride<br>Chloroprene<br>Cresol<br>Dinitrophenol<br>Dioxan<br>Lead<br>Mercury and bichloride of mercury<br>Nitrobenzene<br>Phenol<br>Turpentine<br>Uranium<br>Vanadium | (a) Nephritis<br>Acute<br>Chronic<br>(b) Necrosis of tubules<br>(c) Fatty metamorphosis of kidney<br>(d) Hemoglobinuric kidney | (1) Routine urinalysis<br>(2) Microscopic for cells, casts, etc.<br>(3) Renal function tests<br>(a) Insulin clearance<br>(b) Urea clearance<br>(c) Dilution—concentration<br>(4) Tests for hemoglobin<br>(a) Benzidine<br>(b) Spectroscopic<br>(5) Stain for hemosiderin in macrophages in urinary sediment |

7. Diagnosis must not rest upon laboratory findings alone but rather in combination with the clinical picture.

In Tables I to III* the technical procedure involved in performing the various tests will not be described, since instruction of that type would fall within the province of a laboratory manual. However, for those who wish to know the details of these various tests, a reference list is appended. The purpose of these tables is to indicate the type of test to be requested of a laboratory when seeking evidence of absorption or intoxication.

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*Acknowledgment is made to W. C. Dreessen and R. F. Sievers after whose tables Tables I to III have been modified.

# PART TWO

## CHAPTER VIII

## THE INDUSTRIAL SOLVENTS

Medicine is at present going through a phase of evolution which has few parallels in its history. In the new concept of medicine, attention is focused upon environment as well as upon the patient. The sights have been raised above and beyond the sick bed. The cause for this is dual: sociologic as well as scientific. The sociologic facet of environmental medicine has been indicated in the introduction to this book. At this point, the scientific influence on the future of medicine will be briefly mentioned.

As these pages are written, announcement has been made of an experiment carried out by the General Electric Company, in which their scientists were able to cause a fall of snow by bombarding a cloud with pellets of carbon dioxide. This demonstration presages the production of rain at will and, consequently, man's independence of the whims of nature. No longer do the sciences dominate man: man controls the sciences. However, with each scientific discovery there invariably arises a new danger to mankind which it is imperative to overcome and control. Nowhere is this urgent necessity more evident than with regard to the various industrial chemical processes. Approximately a decade ago the number of chemicals utilized by American industries was relatively small. Today practically all industries use or produce certain chemicals.

Among these various chemicals, the solvents represent a particular challenge to medical knowledge and control. Not only because their name is legion, but also because their number is daily being added to and/or changed to meet new industrial exigencies. Unless the average physician has received special training, he is bound to find himself lacking in a knowledge of chemistry sufficient to diagnose, treat, or prevent the illnesses which are wont to arise from exposure to these solvents. Medical educators should recognize this situation and bend every effort in order to correct the existing curriculum which sends forth graduates with a knowledge restricted to secretion, excretion, and gastric juices.

What is an industrial solvent? What is meant by an aromatic hydrocarbon, and how does it differ chemically from an aliphatic hydrocarbon; in what respect does it differ in its effect upon the human organism? What is a ketone or chlorinated hydrocarbon? To most physicians, organic chemistry is forgotten knowledge. It is therefore the purpose of the author to introduce a discussion of the solvents with an elementary survey of organic chemistry. But for obvious reasons such a review can have only a limited scope, as it is intended solely for the average physician to whom it might prove an aid toward a better understanding of the chemical nature of the industrial solvents. A brief consideration of organic chemistry will also familiarize the reader with the various chemical terms as they appear here and elsewhere in the literature on occupational diseases.

In addition to a consideration of the chemical nature possessed by the industrial solvents, it is indicated to give some thought to the physiologic reaction which they induce, the method of their absorption and elimination, their predilection for certain organs or systems of the body, and the manner in which they might cause death. Experience has enabled us to know that a certain substance under given circumstances will produce fairly typical reactions. The general statement can be made that the solvents or their gases and vapors act as irritants, asphyxiants, anesthetics, or injurious agents upon the blood or blood-forming organs. Within limits the following classification is acceptable:

1. Simple asphyxiants: nitrogen, hydrogen, helium, methane, ethane, nitrous oxide, ethylene, acetylene (Not common industrial incidents)
2. Chemical asphyxiants: carbon monoxide, cyanogen, hydrocyanic acid, cyanogen chloride, acetonitrile, benzonitrile
3. Irritants to the upper respiratory tract: ammonia, formaldehyde, hydrochloric acid, hydrofluoric acid, sulfuric acid, chlorine, bromine, iodine, acrolein, and hydrogen sulfide
4. Irritants to the pulmonary tissue: nitrogen dioxide, phosgene, phosphorous trichloride and pantachloride, arsine, methyl bromide, methyl chloride, and, with severe gassing, bromine, chlorine, and iodine
5. Irritants to the nervous system—anesthetics and narcotics: gasoline, naphtha and benzine (rarely), methyl bromide, methyl chloride, methyl alcohol and other halogenated hydrocarbons; carbon tetrachloride, trichlorethylene, trichlorethane, tetrachlorethane, and other chlorinated hydrocarbones.
6. Poisons which may injure the kidneys and the liver: the halogen compounds of methyl such as methyl chloride, methyl bromide; the chlorinated hydrocarbons such as trichlorethylene, carbon tetrachloride, etc.
7. Poisons which may injure the blood or blood-forming organs: the aromatic hydrocarbons; the nitro compounds

Obviously, not all industrial solvents are listed in this classification, nor was it possible to indicate that certain of them exercise overlapping actions; that is, an ability to affect several systems simultaneously or consecutively. For instance, hydrogen sulfide may affect the lungs but in extreme concentrations the immediate effect is upon the respiratory center, and under these conditions the lungs will usually escape involvement. Furthermore, the accompanying classification does not differentiate between the acute and the chronic exposure.

It is not amiss to point out that many observations by foreign writers regarding the action of industrial solvents are not in agreement with our American experience. In the German literature particularly there is a tendency to make sweeping statements regarding injury sustained from solvent intoxication, without adequate qualification of observations. For example, in an otherwise excellent book, Lehmann and Flury* state, "*All* solvents are more or less *strong* poisons to the nervous system." And in another paragraph one reads, "All solvents are capable of injuring the heart, although in very different degrees. This causes the tissues to receive an insufficient supply of blood and oxygen, disturbed distribution of the blood, engorgement of the venous vascular regions and the blood vessels. Outward signs are the blue coloring of the skin and mucous membranes, cyanosis and dyspnea." In refutation, it is well known that certain solvents may cause cyanosis and dyspnea because of the the limited oxygen-carrying capacity of the blood cells, or a direct involvement of the respiratory center, while they may yet fail to injure the heart itself. Lehmann and Flury infer that chronic exposure to the chlorinated hydrocarbons may produce arteriosclerotic changes; they also infer that angina pectoris may result from trichlorethylene. But such observations are not the experience of the writer and it is very doubtful that they will find corroboration by American toxicologists.

Admittedly there is a need for greatly increased experimentation in the field of industrial toxicology. Sufficient knowledge regarding the action of many chemicals upon the blood, the body metabolism, glandular activity, the cellular structures of the tissues, or the enzymes is still lacking. We need to know more about the changes which occur within the organic cells once a chemical is absorbed; and the oxydative, reductive, or hydrolytic action and the nature of decomposition products are likewise still not sufficiently explored. Industrial toxicology offers a great field of adventure in experimental research.

Organic chemistry is the study of the compounds of carbon. The structure of this element or its compounds is best understood when it is represented in a graphic manner. It will be recalled that in chemistry a structural formula consists of symbols representing atoms and lines or bonds representing valences. The simplest organic compound, methane, is expressed through the following structural formula:

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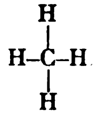
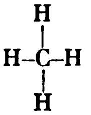
* Lehmann, K. B., and Flury, F.: Toxicology and Hygiene of Industrial Solvents (translated by King, Eleanor, and Smythe, Henry F., Jr.), Baltimore, 1943, Williams & Wilkins Co., p. 52.



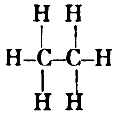
$\underset{\mathrm{H}}{\overset{\mathrm{H}}{\mathrm{H{-}C{-}H}}}$

This structure indicates one atom of tetravalent carbon (four bonds) combined with four atoms of univalent hydrogen. By the successive addition of $CH_2$ to the simplest element, a homologous series is formed, as, for instance:



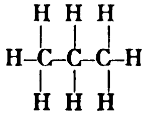
$\underset{\mathrm{H}}{\overset{\mathrm{H}}{\mathrm{H{-}C{-}H}}}$

Methane



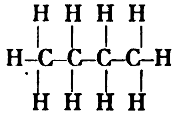
$\mathrm{H{-}\underset{H}{\overset{H}{C}}{-}\underset{H}{\overset{H}{C}}{-}H}$

Ethane



$\mathrm{H{-}\underset{H}{\overset{H}{C}}{-}\underset{H}{\overset{H}{C}}{-}\underset{H}{\overset{H}{C}}{-}H}$

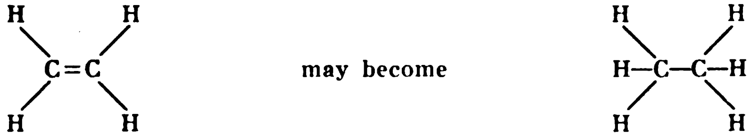
Propane



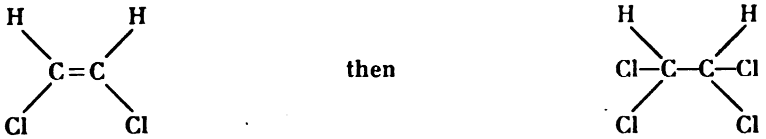
$\mathrm{H{-}\underset{H}{\overset{H}{C}}{-}\underset{H}{\overset{H}{C}}{-}\underset{H}{\overset{H}{C}}{-}\underset{H}{\overset{H}{C}}{-}H}$

Butane

**Saturated and Unsaturated Compounds.**—In the foregoing methane or paraffin series, each carbon atom is joined to an adjacent carbon atom by a single valence bond; that is, each valence is satisfied. Such compounds are said to be *saturated.* There exist other compounds, however, in which one or more of the four valences of a carbon atom remain unsatisfied. These are known as *unsaturated* compounds. Because in the first group there are no unused valence bonds, the atoms are unable to combine by direct addition with atoms of hydrogen, the halogens, etc. But in the second group, because of the unused valences, the carbon atoms will combine with hydrogen, the halogens, and other elements. Such compounds may possess one or more double bonds, = or triple bonds, ≡. Ordinarily, an atom of certain of the elements may be added to each of the two ends of a double bond and either one or two atoms at each end of a triple bond:

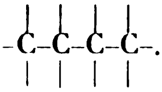


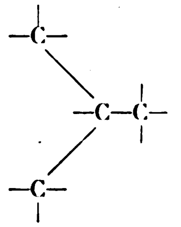
H C C H (acetylene) becomes:



Ethylene dichloride

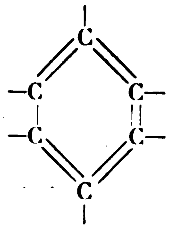
Tetrachloroethane

**Chains and Rings of Atoms.**—If the simple carbon atom, $-\overset{|}{\underset{|}{C}}-$, becomes linked with other carbon atoms, using some of the valence bonds to join them together, a chain of carbon atoms is obtained, as, for instance in  $-\overset{|}{\underset{|}{C}}-\overset{|}{\underset{|}{C}}-\overset{|}{\underset{|}{C}}-\overset{|}{\underset{|}{C}}-$. This simple chain may become branched as in:

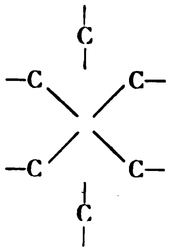


**The Aliphatic Compounds.**—The aliphatic compounds have molecules composed of chains of carbon atoms, either straight or branched, as previously illustrated. The largest group of these is the petroleum hydrocarbon series.

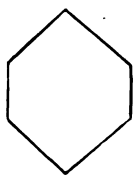
**The Aromatic Compounds.**—The aromatic compounds are characterized by a special arrangement of the carbon atoms in a structure called a ring, the "benzene ring" being the most common industrial example of this hexagonal formation. It is represented structurally in the following manner:



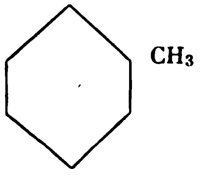
or sometimes as



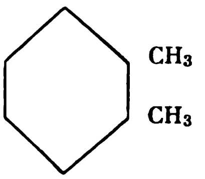
or by



leaving unexpressed the six carbon atoms and any hydrogen atoms which may be attached to them. If a methyl group is added to this structure, substituting it for one of the hydrogen atoms of benzene, $C_6H_6$, toluene is obtained:

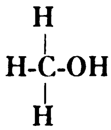


or xylene



by adding two methyl groups

**The Alcohols.**—Alcohols may be regarded as substitution products derived from hydrocarbons, by replacing hydrogen atoms with hydroxyl (OH) groups. Thus methyl alcohol ($CH_3OH$) structurally is



$$\begin{array}{c} H \\ | \\ H\text{-}C\text{-}OH \\ | \\ H \end{array}$$

and is formed by the replacement of a hydrogen atom in methane by OH; ethane becomes ethyl alcohol by the replacement of the H in ethane by OH.

**The Aldehydes.**—An aldehyde is an organic compound having a characteristic group —CHO. Aldehydes are formed from alcohols by partial oxidation. Formaldehyde is the most important member of this group and is produced by passing a mixture of methyl alcohol and air over a hot copper gauze: oxidation of $CH_3OH$ (methyl alcohol) produces HCHO (formaldehyde), or the oxidation of $C_2H_5CH$ (ethyl alcohol) produces $CH_3CHO$ (acetaldehyde).

**Acids and Esters.**—Organic acids contain the carboxyl group, —COOH, in combination with a hydrogen or a radical, as formic acid (HCOOH), or acetic acid ($CH_3COOH$).

The organic acids react with alcohols in the presence of catalysts to produce esters, the hydrogen of the —COOH group being replaced by a hydrocarbon group:

$$\underset{\text{Acetic acid}}{CH_3COOH} + \underset{\text{Ethyl alcohol}}{C_2H_5OH} \rightarrow \underset{\text{Ethyl acetate (an ester)}}{CH_3COOC_2H_5} + H_2O$$

It should be kept in mind that the parent hydrocarbon contributes a definite carbon skeleton, to which elements may be added in chains or in the ring structure; thus the formation of the various compounds, such as those of the nitro group, the chloro group, or the halogenated hydrocarbons is more easily understood.

It is not intended to convey the impression that only intricate industrial processes demand a certain knowledge of chemistry. The industrial physician is frequently confronted by apparently difficult problems which, however, become amazingly simple when the chemistry of the involved substance or ingredients is understood, as illustrated in the following case:

A maid employed in a motor court claimed that she was "overcome by fumes" when she had used a "bowl cleaner" while cleaning out a toilet bowl. In relating the incident she stated: "There were two containers in the bathroom, one labeled Apex Bowl Cleaner, the other Apex Bleach.* I picked up the bowl cleaner and dumped some of it into the toilet bowl. Just then the door bell rang and I went to answer it. When I came back I picked up a container and dumped in some more. When I did that a strong gas arose. It burned my eyes, nose, and throat and I began to cough violently. I could hardly get my breath. A woman in the next cottage phoned different doctors but none could be located. Finally she contacted a doctor in town who advised me what to do for emergency treatment. I did what he told me and continued his treatment for several days. My boss was away for four days. When he came home I reported to him, and he sent me to a doctor. This doctor didn't believe me. He said he used the same stuff in his home and office but it never caused any trouble. He said it was safe."

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* The word Apex has been substituted for the actual trade name.

Under a Public Liability claim this woman entered suit against her employer. She was referred to the writer. To him her story appeared plausible in view of the fact that her description of the incident corresponded perfectly with a condition which could occur in the presence of an irritant gas. She was asked whether she had poured the same substance into the bowl before and after she had gone to answer the doorbell, or whether she might inadvertently have picked up the bleach on the second occasion. The maid admitted the possibility of mixing the contents of the two cans. Whereupon the question then arose as to what the chemical components of these two substances were. Since the two products were manufactured by a local company, the nature of the two ingredients was easily ascertained.

The active ingredient in the first can was sodium hypochlorite ($NaOCl$), while the active ingredient in the second can was sodium bisulfate ($NaHSO_4$). If these two substances were to be mixed, what would be the chemical reaction? Sodium hypochlorite is stabilized by a small amount of alkali, but if such a substance is acidified, chlorine gas is liberated. That is what took place in the case which has been reported here, since sodium bisulfate in solution gives a strongly acid reaction.

This case has been cited as an exemplification of the value of recalling one's knowledge of simple, elementary chemistry. But it also illustrates the contention made in the chapter on The Basic Approach to a Diagnosis of Occupational Disease, that it is necessary to secure accurate information in any given case and then to proceed with consecutive logical deductions. In this particular case it did not seem plausible to assume that the maid was fabricating the syndrome she related, since it suggested an exposure to some type of irritating gas. The next step was to determine whether the two substances she used could, if mixed, give rise to such a gas. Having secured the information as to the chemical nature of the constituents, and appreciating the chemical reaction that could take place, it was apparent that chlorine had accidentally been liberated. Since chlorine is an irritating gas, the claimant's symptoms were explained and the diagnosis established.

If it is essential to have some knowledge of the chemistry of the so-called industrial solvents, gases, and vapors, it is equally important to appreciate the physiologic reactions which they produce: to know how these substances are absorbed, distributed, and eliminated by the body. For such information the writer can do no better than to present the following discussion from the excellent work of Henderson and Haggard:[1]

"Noxious gases and vapors, other than irritants and simple asphyxiants, exert their action only after absorption into the blood. The body is poisoned by the absorbed substance in the same way, and to the same extent, as it would be if a like amount entered the body through any other channel of absorption, i.e., if it were swallowed or were given intravenously or per rectum. The toxic effects of noxious substances are determined by the amount accumulated in the body, i.e., by the total dosage. The idea of total dosage is universally understood in respect to poisons which are swallowed or which are given by injection. It is less well understood in respect to substances absorbed through the lungs. But the fact remains that, with qualifications in respect to the immediate anesthetic effects of volatile hydrocarbons, it is total dosage—the number of milligrams per kilogram of body weight—of the noxious substance, volatile or otherwise, taken into the body which determines the effect produced.

"The obvious, basic feature of total dosage relates the toxicology of noxious gases and vapors to that of all other noxious substances. But it is a feature often overlooked because of the practice of expressing physiological response to gases and vapors only in terms of concentration of the gas or vapor in the air breathed and length of exposure. These values are practical in setting up working standards for ventilation but in them is involved not only total dosage but also the physical characteristics of the gas or vapor, particularly its solubility, its molecular weight, if the concentration is expressed in volume relation to vapor, and the peculiarities of absorption through the lungs. The concentration and length of exposure which lead to accumulation of a certain amount of one gas or vapor in the body may lead to the accumulation of a widely different amount of another gas or vapor. Representations of effect in terms of concentration and exposure have therefore only empirical significance unless the variables involved and their relation to each other are understood.

"*Maximum Possible Absorption.*—There are two obvious general facts in regard to the absorption of any volatile substance through the lungs: (1) No more of the substance can be absorbed than is taken into the lungs in the air breathed; and (2) absorption ceases when an equilibrium is reached between the amounts *in the body* and *in the air.* The second of these will be dealt with later. The first—the maximum possible absorption—will be considered here only as it affords a rule of thumb which is useful in making approximations of the physiological response to be expected from various concentrations of a gas or vapor in the air breathed.

"The amount of any vapor brought to the lungs for absorption is determined by the product of three factors: (1) the volume of pulmonary ventilation per minute; (2) the concentration of the vapor in the air expressed in milligrams per liter; and (3) the length of exposure. The pulmonary ventilation of a man of average size performing work requiring moderate exertion may be taken as 8 liters per minute, or, in round numbers, 4,000 liters in an 8-hour working day.

"If the air breathed contains 1 mg. per liter of some volatile substance the amount brought to his lungs, and hence the maximum amount that can be absorbed, is 4 g. The same maximum dose would be accumulated in an exposure of 1 hour to a concentration in the air 8 times as high as that given.

"The next question would be that of the pharmacological effect of such a dosage. It is often possible to obtain from medical literature some idea of the toxic or non-toxic doses for single and repeated administrations of many of the volatile substances used in industry. The relation of dosage and physiological effect has a much more definite meaning than does that of concentration in the air and physiological effect.

"At the commencement of inhalation of any gas or vapor, that portion which is taken up by the blood is carried in the arterial stream to the capillaries, where exchange of the gas or vapor takes place by diffusion between the blood and tissues. Here, as in the lungs, a nearly complete equilibrium is reached almost instantaneously. The venous blood, therefore, leaves the tissues with a concentration of gas or vapor practically identical with that developed in the tissues through which it has passed. The blood, with a reduced amount of the gas or vapor, then returns to the lungs, where it is again brought into equilibrium with the concentration of gas or vapor in the lungs. As the process continues, the amount of gas or vapor, and hence the tension, constantly rises in the tissues. The amount in the venous blood also rises correspondingly and tends gradually to approach that in the arterial blood. More and more of the gas or vapor is thus carried back to the lungs by the venous blood; consequently a decreasing amount is taken up by the blood from the air in the lungs and by the tissues from the blood. The limit approached is a state of saturation—equilibrium—at which absorption ceases (except to replace the small amounts eliminated in urine and sweat) and the body is saturated with the particular gas or vapor at the concentration existing in the air breathed. At this saturation the body will contain an amount of gas or vapor equal to the product of three factors: (1) the weight of the body; (2) the concentration of the gas or vapor in the air in the lungs; and (3) the coefficient of solubility of the gas or vapor in the body tissues at body temperature.

"*Equilibrium Value.*—What we have called here the equilibrium value—the full saturation of the body for any one concentration of gas or vapor—is of great practical importance: it expresses completion of absorption and therefore does away with all further consideration of time of exposure in relation to concentration of the gas or vapor in the air. If the total amount of gas or vapor that can have been absorbed into the body at equilibrium with any given concentration in the air causes no harm, then that concentration may be inhaled indefinitely, since at equilibrium no more of the gas or vapor can accumulate in the body. In this respect absorption by way of the lungs differs from absorption through any other channel. When a noxious substance is repeatedly swallowed, it continues to enter the body as long as it is taken; when a gas or vapor is inhaled, the absorption stops when the tension of the gas in the body corresponds to that outside of the body. Both the amount in the body and the concentration in the blood are then stationary at their maximum for the particular concentration in the air.

"The equilibrium value thus establishes a limit for maximum possible absorption. With highly soluble gases or vapors, absorption may continue throughout an entire working day, but with those of low solubility, equilibrium is reached within a period of less than 8 hours and absorption is terminated.

"In discussing maximum possible absorption an example was given in which a man was exposed to air containing 1 mg. per liter of vapor; he breathed 4,000 liters in 8 hours and his maximum possible absorption was 4 g. The saturation or equilibrium value for any gas or vapor—that is, the maximum amount that can be absorbed at any concentration—is determined as the product of the weight of the body, the concentration of the gas or vapor in the lungs, and its coefficient of solubility. A man of average size weighs about 70 kg.; he will be assumed here to breathe an atmosphere containing ether vapor; the coefficient of solubility of ether is 15. The saturation value for such a man breathing an atmosphere containing 1 mg. of ether vapor per liter of air would be 1.05 g. He could not accumulate a greater amount in any length of exposure, no matter how long. The saturation value in a gas or vapor of this solubility and lower becomes, therefore, a primary consideration in determining the maximum possible absorption.

"The calculations made here are valid only for vapors which are essentially non-reactive in the body, as are the great majority encountered in industry. With reactive vapors, such as ethyl alcohol, the continual oxidation would keep the dose much lower than that calculated as above and saturation could not be attained. Such reactivity is a distinct advantage when the products of oxidation are, as with ethyl alcohol, non-toxic. However, it is a disadvantage when, as with aniline, the oxidation products are toxic. The continual oxidation prevents attainment of saturation and hence of cessation of absorption; the continued absorption leads to progressive accumulation of the toxic products of oxidation.

"*Concentration of Gas or Vapor in Blood, Urine and Expired Air.*—The concentrations of vapor in the blood, urine and alveolar air bear definite relations to one another and to the amount of vapor absorbed in the body. But to use these concentrations for more than an approximate estimation of the amount absorbed, or to compare physiological effects, the factors affecting these relations must be taken into consideration. It is only when absorption has ceased, or is at an extremely low rate and a virtual equilibrium of the vapor throughout the body has been reached, that the concentrations of the blood, urine or alveolar air become precise indices of the amount in the body. Short of this equilibrium, and while absorption is continuing actively, the concentration in the arterial blood is at a continually higher level than that in the venous blood. Furthermore, the blood drawn from veins in different parts of the body during this time may show widely different concentrations. With slow absorption from low concentrations of vapor inhaled, these differences are slight; with rapid absorption from high concentrations, they may be large.

"The maximum amount of the inhaled gas which any organ or tissue can absorb at a given tension is determined by the solubility of the gas in that tissue. The rate at which the gas accumulates in the particular tissue depends both upon the solubility of the gas in that tissue and also upon the amount of the gas brought to the tissue in any period of time. The latter factor is determined by the blood supply in relation to the bulk of the tissue. Thus a tissue with a comparatively low solubility for the gas and a large blood flow approaches saturation rapidly, whereas a tissue of high solubility and small blood flow approaches saturation much more slowly.

"During bodily rest it may be estimated that approximately 80 per cent of the blood passes through 20 per cent of the tissues in each round of the circulation; the remaining 80 per cent of the tissues receive only 20 per cent of the blood. Bone and fat, which together constitute a large part of the tissues of the body, receive a comparatively small blood flow. The flow to the brain, viscera and glands is large. This latter group of organs therefore approaches saturation at a much more rapid rate than do the bone and fat. The same relation holds true of elimination.

"The rate of saturation of tissues with a large blood supply follows closely the concentration of a gas or vapor in the arterial blood. The concentration in these tissues therefore rises at first much more rapidly than in the tissues with a smaller blood supply, but the less rapidly saturating tissues act as a buffer to prevent immediate and complete equilibrium. This is the case both during absorption and elimination of the gas. During absorption the tension of a gas in any tissue cannot rise above the tension of gas in the arterial blood, nor can it fall below this limit during elimination. The tissues of small circulation and slow saturation and desaturation influence the concentration in the venous blood and consequently that in the lungs and arterial blood also.

"Certain gases and vapors, particularly the anesthetic hydrocarbons, exert their acute action specifically upon one organ—the brain—and are largely inert in regard to others. The brain has an extremely large blood supply; the anesthetic action therefore tends to follow closely the concentration in the arterial blood and is little influenced by the total amount absorbed. The concentration in the peripheral venous blood indicates only the degree of saturation in the particular organ from which the blood is returning. Because of the large circulation to the brain the concentration in the brain builds up rapidly and the concentration in the blood from the jugular vein follows closely that in the arterial blood. Because of these relations the momentary inhalation of a high concentration of an anesthetic hydrocarbon, e.g., ethyl ether, may cause marked symptoms before any large amount has been absorbed into the other parts of the body. On breathing fresh air the effects pass off equally rapidly since, when the inhalation of the vapor ceases, there is an immediate fall in the concentration in the arterial blood and correspondingly in the brain. If the inhalation has been prolonged with the accumulation of a large amount of the vapor in the body, this rapid fall does not occur, since the concentration in the arterial blood is maintained by the vapor returned in the venous blood; a much longer time is then required for recovery.

"The concentration of any vapors in the alveolar air corresponds to that in the arterial blood leaving the lungs at the time the alveolar air is collected. The relation of the concentrations in the air and blood is that of the coefficient of distribution for the particular vapor.

"The concentration of the vapor in the urine corresponds closely to that in the arterial blood passing through the kidneys at the moment the urine is secreted. It must be borne in mind that the urine which has collected in the bladder does not show this relation for any one time; it is instead a composite of the urine secreted during the entire period in which it has collected and the varying concentrations in the blood at these times. The relation between the concentrations in the blood and urine—secreted at one time—is in the same relation as the solubility of the vapors in the two fluids; the difference in these solubilities is usually slight.

"*Elimination of Gases and Vapors.*—Gases and vapors are eliminated largely through the lungs and to a much lesser extent through the urine. Elimination through the lungs follows definite principles which are similar to those of absorption in reverse.

"At the termination of absorption the body contains an amount of the gas which is equivalent to full equilibrium with some tension of the gas, although this may be much lower than the inhaled concentration. If none of the gas is eliminated, the body forms a closed and static system in which the gas is distributed to the various tissues and to the air in the lungs in proportion to their capacity to dissolve the gas. The concentrations in the arterial and venous bloods, under these circumstances, are the same. When elimination begins and part of the air in the lungs is replaced by fresh air, a part of the gas is carried away in the expired air and the concentration of the gas in the arterial blood leaving the lungs is reduced below that in the venous blood coming to the lungs.

"*Accumulation of Gases and Vapors.*—Under industrial conditions exposure to a gas or vapor is limited to the working hours. The time spent away from work is usually more than adequate to permit complete elimination of the gas or vapor and consequently none is present in the body at the beginning of the next daily exposure. There is therefore no accumulation of gases and vapors from daily exposure. The only possible exception to this general statement would be with highly soluble and non-reactive substances, such as methyl alcohol, or with reactive gases or vapors whose decomposition products were eliminated slowly.

"*Reactive and Non-Reactive Gases and Vapors.*—The gases and vapors which exert their action after absorption into the blood may be divided into two classes according to whether they are reactive or non-reactive.

"Reactive gases and vapors are altered within the body and are eliminated to a large extent in forms other than those in which they are absorbed. The toxicological action may be exerted either by the substance in its original form or by the product of such reaction. Ethyl alcohol is an example of a reactive vapor which exerts its action before its destruction has taken place; aniline is an example of a vapor which exerts its main action in the product arising from its reactions in the body.

"Non-reactive gases and vapors are not altered to any appreciable extent in the body and are eliminated in the same form in which they are absorbed. The aliphatic hydrocarbons are examples of non-reactive gases and vapors."

The chemicals which fall under the heading of this section of the book have at different times been variously classified, but probably no one method will satisfy all purposes or the critical opinion of all readers. These substances could be arranged according to the physiologic reaction which they induce, i.e., the irritants, the asphyxiants, the anesthetics, etc.; or as to the pathologic effect upon the nervous system, the blood forming organs or upon such special organs as the liver or kidneys. For the purpose of this book, however, these chemicals will be arranged according to their chemical composition. Under each division there will be named the more important members of the group, followed by a description of the clinical picture that could result from an undue exposure to any one of them. It is obviously impossible to deal with each chemical individually. For instance, under the heading of chlorinated hydrocarbons, some twenty or more substances could be named, but only a few will actually be described. But the reader is asked to bear in mind that as a general rule and with few exceptions, all other members of a given group may be expected to act upon the body in a manner similar to those of the group which have been described in detail, albeit the degree may vary.

### References

1. Henderson, Yandell, and Haggard, Howard W.: Noxious Gases, New York, 1943, Reinhold Publishing Corporation.

# CHAPTER IX

# THE ALIPHATIC HYDROCARBONS

## PETROLEUM SERIES

When, on a sultry Saturday afternoon in August, 1859, oil bubbled near the top of Colonel Drake's well near Titusville, Pennsylvania, a new industry was born, the importance of which he could hardly have dreamed of. For the early "teakettle" refineries, kerosene was commercially the most successful product and, ironically, gasoline was looked upon as a nuisance.

The paraffins of the petroleum series are methane, ethane, propane, butane, pentane, hexane, and octane. Crude oil pumped from the wells contains all the hydrocarbons of the methane series. At first distillation, ethane, propane, and butane pass off and are liquefied by compression. The chief hazard inherent in these gases is their explosiveness. The olefines are ethene, propene, butene, hexene, ethyne, and propyne. While these substances possess narcotic properties, they will cause no harmful effects except in high concentrations. Butadiene, a diolefine, will be mentioned in the discussion of synthetic rubber.

The principal fractions of petroleum on distillation are petroleum ether, petroleum benzine, naphtha, benzine, and gasoline. Naphtha and benzine act upon the nervous system in a manner similar to gasoline but are less volatile and therefore not readily inhaled. Any injury due to undue exposure is usually short and without complications.

When the early Fords, Haynes, and Duryeas were first made, their fuel was a straight-run gasoline and, as such, its action upon the physiologic system was slight. But with a car in every garage, the petroleum industry had to obtain a method of production which would give a better yield; and apart from that, the modern engines were designed for a more volatile type of gasoline. The result was production of a substance containing higher percentages of aromatic hydrocarbons and naphthenes, which means that incidentally the harmful potentialities of the fuel were greatly increased.

In the process of cracking, the heavier molecules of crude petroleum are split into smaller ones suitable for engine fuel. Molecules are divided by cracking, added by polymerization and alkylation, and subtracted by dehydrogenation. By such techniques, gasoline becomes a product made to specifications with but little relation to the original crude oil. In the original cracking process only pressure was used to split the hydrocarbons, until it was found that catalyst action would produce a high octane gas. The Fluid catalyst uses a very fine powder, the Houdry process a pellet, and the Bead process a fine bead. Alkylation utilizes what were formerly waste gases which, with the aid of sulfuric acid, are combined to form molecules with the properties of iso-octane, the perfect antiknock gasoline. In giving a gasoline a rating, it is therefore compared with iso-octane, which is accorded a rating of 100. Any gasoline possessing 77 per cent antiknock efficiency of iso-octane is given an octane rating of 77. This information may help a physician in determining the degree of potential danger whenever the rating of the gas is known.

## Gasoline

**Occupational Exposures.**—Since the use of gasoline is so ubiquitous, an attempt to enumerate or distinguish all occupations employing gasoline would be futile. Because of the proper measures instituted for its safe handling and processing, the petroleum industry shows a lower incidence of intoxications than those which are occurring in other industries. It is more particularly in occupations where gasoline is used without regard for its possible harmful consequences that illness is likely to result. One potential source of danger not frequently recognized exists where gasoline is used as a diluent or thinner, as pointed out by Sterner.[1]

**Pathology.**—Intoxication is invariably the result of inhalation. Absorption through the skin is highly questionable. Injuries resulting from a combination of swallowing and inhaling have been reported by several observers[2-5] in other countries, and the author reported the only case[6] in the American literature of pleural effusion resulting from such an accident. More recently an almost identical case was reported by Cope[7] in England.

According to Machle[8], the pathologic changes induced by gasoline are dependent upon its irritant action and its lipolytic activity. Moderately severe to severe absorption will produce various types of pulmonary inflammation such as bronchitis, pneumonitis, bronchopneumonia, petechial hemorrhage, and pleural effusion.

Severe exposure to a high octane fuel may damage the central nervous system, resulting in edema of the brain, petechial hemorrhages, and myelitic changes. However, this problem has not yet been sufficiently studied to allow postulation of positive findings. Animal experimentation has shown a certain involvement of the liver and kidneys, but the writer has not observed similar changes in the human being.

**Signs and Symptoms.**—These are dependent upon the dosage to which the individual has been subjected. In the ordinary handling of gasoline no detrimental effects are noted, as testified by the thousands of gasoline station attendants. If an exposure occurs just above the maximal limits of safety, a mild reaction characterized by headache, drowsiness, and lassitude will result. As concentration and absorption increase, euphoria, excitement, disorientation, a reduced level of consciousness, convulsions, and coma may ensue. Fortunately, such cases are extremely rare, and, when they do occur, the place is always some confined space such as the inside of a tank. Rudd[9] reports severe intoxication among workers who, without protection, had unloaded ships in which leakage from drums was so great that gasoline had been spilled in the holds to a depth of two or three feet. Some of the men were overcome, while others were observed to leave the ships singing uproariously as if they were drunk; yet acute alcoholic intoxication was excluded as a cause.

As for chronic gasoline intoxication, its occurrence must always be regarded as questionable. For a period of twelve years, Machle[8] had 2,300 refinery workers under observation, without ever noting any evidence of gasoline poisoning. They also report negative findings among large numbers of station attendants, tank wagon drivers, and garage mechanics.

**Laboratory Findings.**—No specific laboratory findings can be considered as characteristic. Any change in the blood picture must be attributed to hemorrhage, or to some aromatic hydrocarbon contained in the mixture. Askey[10] reported a case of purpura hemorrhagica and profound anemia, the cause of which was puzzling until it was learned that the man had been washing metal parts in a gasoline bath consisting of more than 20 per cent benzol. Blood chemistry studies reveal no significant changes unless the intoxication has been extremely severe, at which time there may be a slight increase in the urea and total nitrogen; albumen may also be present in the urine. It is advisable to consult chest x-rays, for they may reveal pathology of varying degrees. The cerebral spinal fluid does not undergo changes.

**Disability.**—An average case of acute intoxication from gasoline will recover within a few hours or a few days, without suffering sequelae similar to the effects from most volatile or anesthetic gases. However, if hemorrhage into the brain or lipolytic action upon the nerve tissue should have resulted, permanent sequelae could be incurred. In such instances one should also suspect exposure to the presence of some complicating poison, such as carbon monoxide. At this point the writer wishes to draw attention to an error which has been written into the literature. Machle,[8] in referring to my case of pleural effusion from gasoline inhalation, stated that the patient subsequently developed coma and convulsions. This is an error: the patient recovered completely.

**Treatment.**—

ACUTE CASES.—If the patient survives the exposure, no specific treatment is necessary.

THE UNCONSCIOUS PATIENT.—If the patient is seen while unconscious, removal to the fresh air and inhalation of from 5 to 7 per cent carbon dioxide in oxygen are indicated. Saturated clothing should be removed and the skin cleansed of any of the material. The patient should be wrapped in blankets and kept warm. At this time *circulatory failure*, if present, should be treated by any of the following drugs: coramine (1.5 cc.), caffeine sodium benzoate (7½ grains; 0.5 Gm.), metrazol (1½ to 4½ grains; 0.1 to 0.3 Gm.) or camphor in oil (1 to 2 cc.) intramuscularly. Coramine and metrazol may be used intravenously and will also act as respiratory stimulants. Artificial respiration is usually felt to be contraindicated because of the existing lung damage, and adrenalin administration has been found to give poor results.

PREVENTION OF PULMONARY COMPLICATIONS.—The lungs should be watched for complications, such as bronchopneumonia. After the first portion of the acute stage where respiratory stimulation chiefly is needed, oxygen therapy may be substituted for the carbon dioxide and oxygen inhalations. For cough, codeine phosphate or sulfate (½ to 1 grain; 0.0325 to 0.065 Gm.), perhaps in a cough syrup vehicle, is useful.

NERVOUSNESS AND RESTLESSNESS.—These conditions may require sedation by any of the following drugs: chloral hydrate (10 to 30 grains; 0.65 to 1.95 Gm.), phenobarbital (1½ grains; 0.1 Gm.), sodium bromide (10 to 30 grains; 0.65 to 1.95 Gm.), or paraldehyde (2 to 4 drachms; 8 to 16 cc.). The paraldehyde may be mixed with elixir lactate pepsin, poured over crushed ice, and administered by mouth, or it may be given rectally in olive oil with 1.5 cc. benzyl alcohol. Epsom salts (1 ounce; 30 Gm.) should also be administered.

Venesection followed by blood transfusion has been advocated to rid the body of methemoglobin. When ingestion of the substance has occurred, lavage with Epsom salts solution is of value.

CONJUNCTIVITIS.—Due to irritation from petroleum distillate fumes, conjunctivitis may be treated by dropping 1:1000 adrenalin solution into the eyes four times a day, followed by the use of cold applications for from ten to fifteen minutes. Bland boric acid ophthalmic ointment should also be used.

CHRONIC INTOXICATION.—As previously stated, the writer questions the occurrence of chronic intoxication, but with the addition of the aromatic hydrocarbons to present-day gasoline, the development of anemia is possible. In such a case, the patient should be removed from his occupation. The *diet* should be high in calories with added vitamin preparations. For *secondary anemia*, ferrous sulfate (5 grains; 0.32

Gm.), two tablets three times a day, may be used. The ferrous sulfate preparations seem effective in relatively small doses, and they also seem to cause less gastrointestinal discomfort than other iron compounds.

**Illustrative Cases.**—

CASE 1.—A white man, aged 31 years, was brought to the Golden State Hospital complaining of severe chest pain; he also had dyspnea and cyanosis. His employer who accompanied him stated that several hours previously the patient was engaged in buffing an auto fender. Desiring some gasoline to remove the fine dust particles, he attempted to siphon gasoline from the tank of the car. Inserting one end of the "airline hose" into the tank, he stretched out on the floor below and started to draw. Due to the diameter of the hose, the worker had difficulty in removing the hose from his mouth when the gasoline gushed through. This time interval and the patient's position enabled a large quantity of gasoline to enter the patient's mouth. By the time the victim got to his feet he was strangling violently. He stated: "All I spit out was fumes." His mouth and throat were burned, and within a half hour he experienced pain in the right chest, especially around the nipple area. This became so distressing that he was taken first to the Georgia Street Receiving Hospital, and then to our clinic.

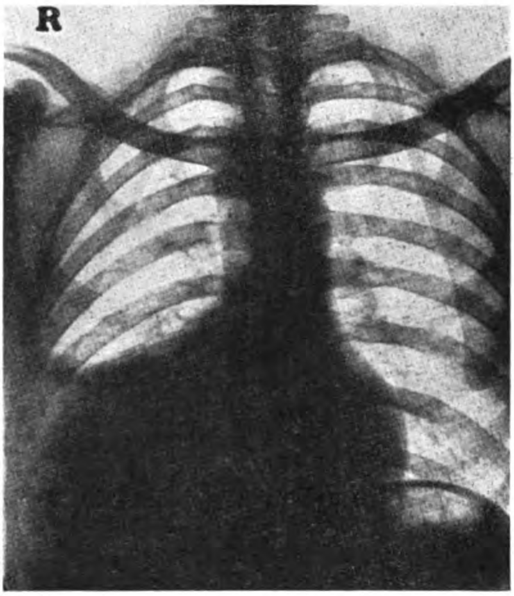

Fig. 2.—May 20, 1937. Films taken fifty-two hours after exposure show fluid level in right chest with some clouding of left lung. (Johnstone: Indust. Med. 7: 243-251 [May], 1938.)

*Physical Examination.*—Examination revealed ashen-gray facies with general cyanosis. He assumed a sitting position, leaning to the right, and refused to lie down, claiming that his breath would be cut off. Respirations were 32 and shallow; pulse, 90 with fair volume; temperature, 100; blood pressure, 102/66. Examination of the chest revealed a pronounced lag on the right; percussion sounds on the left were slightly less resonant than normal; on the right they were noticeably diminished. There were scattered râles throughout both lungs. The

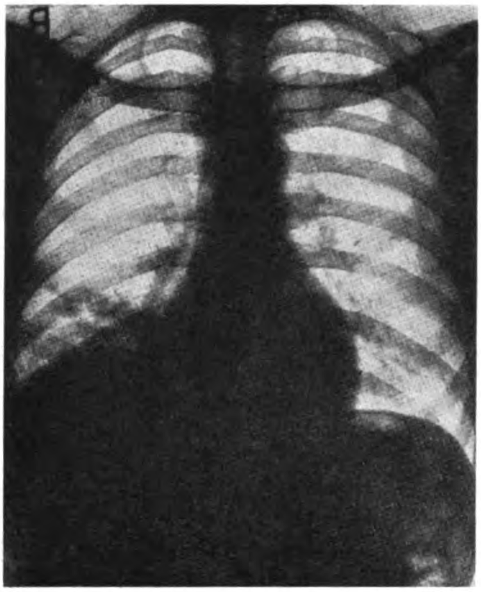

Fig. 3.—May 24, 1937. Films taken four days later show partial recession of fluid level with cloudiness existing above fluid level. (Johnstone: Indust. Med. 7: 243-251 [May], 1938.)

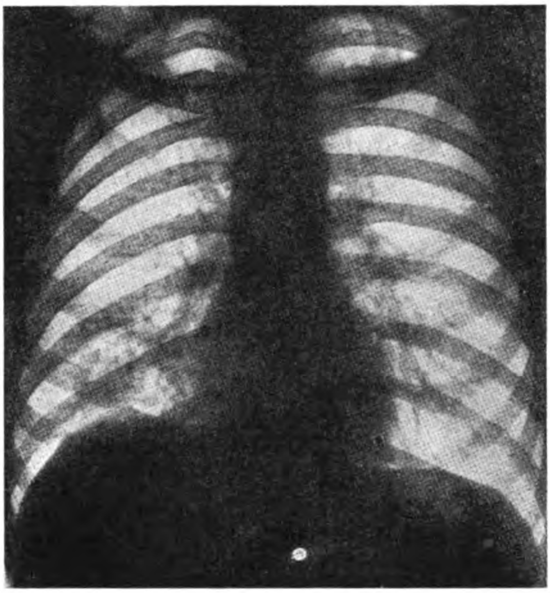

Fig. 4.—May 27, 1937. Greater recession of fluid level and more normal appearance of right lung field. (Johnstone: Indust. Med. 7: 243-251 [May], 1938.)

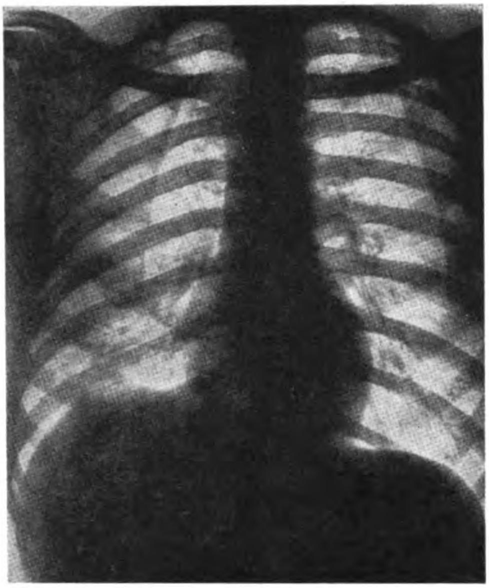

Fig. 5.—June 3, 1937. Disappearance of evidence of fluid. Evidence of pleurodiaphragmatic adhesion. (Johnstone: Indust. Med. 7: 243-251 [May], 1938.)

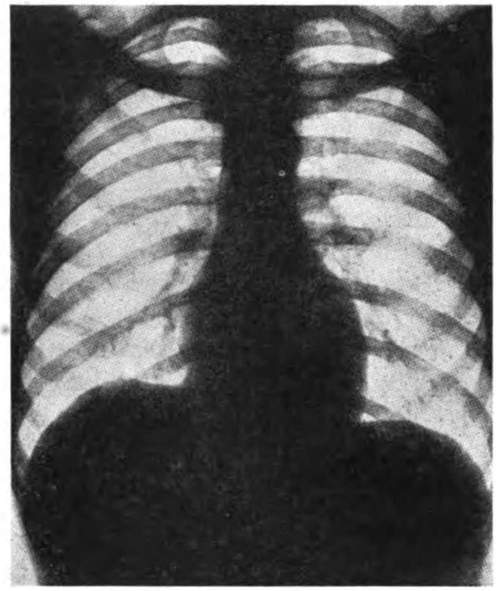

Fig. 6.—June 11, 1937. Lung fields in accord with the normal, well-aerated lungs. Adhesion now reduced to a very small area. (Johnstone: Indust. Med. 7: 243-251 [May], 1938.)

rest of the physical examination and the details of the progress of the case will not be given at this time. He developed a hacking, harassing cough which was productive of a brown sputum. Within thirty hours, evidence of a pleural effusion existed. Roentgenograms confirmed this suspicion. Further confirmation was obtained by aspiration of 10 cc. of amber-colored fluid. The patient made an uneventful recovery, and was discharged on the sixth day (Figs. 2 to 6).

*Laboratory Examination.*—The laboratory study on this patient was entirely negative. The urine showed a trace of albumin, the blood chemistry was normal, and the sputa revealed no evidence of tubercle bacilli and only a few scattered pneumococci. Examination of the sample of effusion showed it to be reddish-yellow and slightly cloudy. Microscopic examination revealed numerous lymphoid and blood cells. Inoculation of various media revealed no growth, and the guinea pig inoculation produced no pathology in two months.

*Discussion.*—Testimony of the employer and a fellow employee as eyewitnesses to the accident was obtained. The patient had been in good health, had lost no time from his work for years, and personal and family history denied the presence of tuberculosis. A sterile effusion argued against the possible etiology of acute infection. The suddent onset and the rapid return to normalcy were in favor of the alleged cause. The patient made an uneventful recovery without sequelae.

CASE 2.—A movie director engaged three men to renovate a yacht he had just purchased. One man was assigned to clean and polish the engine room, which was a rather small, confined place. An unidentified gasoline was employed in removing the grease. Failing to appear for lunch, one of the men was found on the floor of the engine room in a stupor. The glass bottle which contained the gasoline was broken, and the room reeked of gasoline. Brought to the deck, the patient regained consciousness but complained of severe frontal headache and smarting of the eyes. Medical examination two hours later found no objective disturbance, and the laboratory findings were negative. He alleged headache and dizziness for a period of five weeks, at which time he was denied further compensation. Four days after compensation ceased he returned to work.

## Tetraethyl Lead

**Toxicity.**—The addition of tetraethyl lead to gasoline results in a noxious substance, the hazard of which is confined chiefly to those engaged in blending or producing this type of motor fuel. In 1924 a number of deaths occurred among workmen at plants where this fuel is manufactured. As a result, a special committee investigated the toxicology of tetraethyl lead and concluded that:

1. Tetraethyl lead constitutes no undue hazard to the garage worker, gasoline pumper, or the general public.

2. A relatively small industrial group is actually exposed to the hazard. Of this group, the chief ones are those who produce or blend this substance or those who come in contact with its products of decomposition, such as cleaners of storage tanks or railroad tank cars.

3. Minute amounts spilled upon the skin in handling the liquid produce no ill effects, since the quantity of this substance per gallon of gasoline is so low as to be toxicologically insignificant.

4. The preventive problem can be reduced to the minimum by requiring tetraethyl lead to be colored by a dye to distinguish it from other types of gasoline, and by having it properly labeled wherever it is sold, pumped, or stored.

**Pathology.**—The pathologic changes which occur are widespread but are not specific of this poisoning. Congestion of all the viscera is common, as well as hemorrhage into all parts of the body, although hemorrhage into the gastrointestinal tract has not been noted in man. The greatest amount of hemorrhage occurs in the white matter of the brain, together with engorgement of the cerebral vessels. The next site to be most affected are the lungs, where hemorrhage, congestion, and edema are found. Degenerative changes in the liver, kidneys, heart, spleen, and pancreas have been reported.

**Symptoms and Signs.**—These vary with the degree of exposure. They appear within a short time in one severely exposed, while in the one who has received a small dose, symptoms are usually delayed. This, according to Machle,[11] is the result of two factors: (1) "The decomposition of lead within the body with the liberation of water-soluble lead compounds; and (2) the delayed absorption from either the pulmonary epithelium or the skin."

**Prodromal Period.**—Investigators of this type of intoxication state that there is a prodromal period of from eighteen hours to eight days prior to the onset of real symptoms, varying inversely with the magnitude of the exposure and the severity of the poisoning. The victim appears to become increasingly nervous and sleep is difficult and disturbed by terrifying dreams. Apprehension and anxiety progress into states of excitement accompanied by headaches and vertigo. Occasionally the patient becomes depressed and evidences delusions of various types. Generalized weakness and fatigue are common complaints.

**Physical Examination.**—Upon examination the reflexes are hyperactive, although those reflexes not normally present are not seen. The eyegrounds reveal no abnormal findings, although visual weakness may be complained of, because of weakness of the extrinsic muscles of the eye. Tremor, especially of the upper extremities, is noted in the majority of the severely exposed. The heart rate is slow and the systolic and diastolic blood pressure is lowered. Laboratory studies yield little diagnostic significance.

**Complications, Sequelae, and Convalescence.**—What is the end result in patients with nonfatal tetraethyl lead intoxication? Machle[11] studies a series of cases over a long period of time. He states:

> "In mild and moderately severe cases in which there were brief periods of delirium no complications developed. As a consequence of circulatory weakness

and exhaustion in severe or fulminating cases a variety of complications may conceivably develop, such as acute cardiac dilatations, cerebral edema, pulmonary edema or similar lesions associated with terminal states. No complications occurred in the present series, however.

"Sequelae have not been seen. Convalescence may be protracted with symptoms persisting as long as from eight to ten weeks, but no residual symptoms or evidences of damage have been observed. A group of fourteen patients not included in this series have been under medical observation at monthly intervals continuously since the time of intoxication in 1925 and have not given any evidence of subnormality, physical alteration or increased susceptibility to intoxication. They have been continuously at work in the manufacturing operation, the majority of them having been in supervisory positions during the past two years where the exposure is often greater than that experiencd by day workmen. Such evidence, plus the absence of focal brain lesions at necropsy, indicates the reversible nature of the injury from tetraethyl lead and reduces the likelihood of damage from cumulative subclinical exposures."

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# CHAPTER X

# THE ALIPHATIC HYDROCARBONS—Cont'd

## The Alcohols

The alcohols methyl, ethyl, propyl, butyl, and amyl belong to the anesthetic group of gases, but in addition to their anesthetic action they all, and particularly methyl, exercise a direct poisonous effect.

**Methyl alcohol** ($CH_3OH$), also called methanol, is used as a solvent in the preparation of varnishes and lacquers, in the manufacture of some synthetics, in resins and stains, and as a denaturant for ethyl alcohol. Methanol has a disagreeable odor and taste, due to the presence in crude methyl of acetone, ethyl methyl ketone, and certain acetates.

As typical of the anesthetic gases, it chiefly damages the nervous system, particularly the optic nerve, and may result in blurred vision, pain in the eye, and partial or even permanent blindness. After massive dosage, degenerative changes in the kidneys, the liver, and brain have been reported. There is no evidence that exposure causes any disturbance of hematopoiesis. The toxic properties of methyl alcohol have been ascribed to its oxidation products, formaldehyde and formic acid, or to the unoxidized methanol itself.

It is important to note that in equal concentrations, methyl alcohol is not as toxic to the body as ethyl, although its effect is much more harmful because it is only very slowly eliminated. For this reason, accumulation can take place. Whereas a single exposure might cause no disturbance, repeated dosage of small amounts gradually builds up within the body to result eventually in intoxication. This reaction is somewhat comparable to that of methyl bromide, as we shall see later on. Hygienists and safety engineers should appreciate the factor of accumulation and should be cautious in placing too much emphasis upon the concentration within the working environment, while neglecting to carry out repeated examinations of the blood, urine, and expired air of the workers for evidence of the possible presence of methyl alcohol.

Fatal poisoning from methyl alcohol is rare in industry. Greenburg and associates[1] made a study of the fusing process where both methanol and acetone were used. Most of the workers had been exposed for a period of over one year, and many of them for two years. Except for a slight loss of weight in one or two instances, the physical examination resulted in normal findings; the ocular fundi were normal and no visual disturbances were noted. Likewise, no sensory or motor alterations from the normal had occurred, and, with a few minor exceptions, the blood pictures were not disturbed. The concentrations of acetone and methanol were still within the approved limits.

**Ethyl alcohol** ($C_2H_5OH$) is widely used as a solvent and as a starting point for the preparation of many compounds. Its action on the central nervous system is more depressant than that of methyl, but since it is rapidly excreted, it remains in the body only a short time. It is equally important that it is not broken down within the body to form noxious compounds. Exposure may cause irritation to the mucous membranes, headache, dizziness, lassitude, lack of concentration, or inebriation. Serious effects are rare.

**Propyl alcohol** ($C_3H_7OH$) and its isomer, isopropyl alcohol ($CH_3$ (2) CHOH), are used as solvents for resins as well as in other chemical procssses. They do not cause harmful effects since they possess only low volatility.

**Butyl alcohol** ($C_4H_9OH$) is usually employed in conjunction with other solvents, and as a reactivator for synthetic resins. While its toxic effects are not clearly defined, it is not known to cause systemic injury when it is used in normal ranges of concentration. However, it is not free from irritating properties. Tabershaw, Fahy, and Skinner[2] examined six plants engaged in the manufacture of ponchos, raincoats, and sleeping pads for military use. They observed that due to either butanol alone (which is normal butyl alcohol) or to a combination of butanol with other solvents, there were complaints of (1) irritation to the eyes, leading to a particular type of corneal inflammation, (2) disagreeable odor, (3) slight headache and vertigo, (4) dermatitis of the fingers and hands, and (5) slight irritation of the nose and throat.

According to these observers, concentrations below 50 p.p.m. do not produce important effects. Above this range, however, ocular disturbances occur. No systemic effect is to be expected in concentrations of less than 100 p.p.m. The cases of dermatitis usually proved to be restricted to a minor fissured eczema located chiefly around the fingernails and along the sides of the fingers.

Cogan and Grant[3] report an unusual type of keratitis associated with exposure to N-butyl alcohol. The lesions were characterized by numerous translucent vacuoles situated in the superficial layers of otherwise normal corneas. The symptoms consisted chiefly of pain and epiphora, which proved to be most severe when the patient first opens his eyes on awakening. The investigators noted these lesions among "raincoat cementers." Keratitis was observed in workers otherwise free of symptoms. The lesions improved spontaneously when the worker was removed from this exposure.

**Amyl alcohol** ($C_5H_{11}OH$) is considered very toxic but little is actually known about its action or course in the body. Because of its low solubility, industrial poisoning from amyl alcohol is not likely to occur. It is irritating to the eyes and upper respiratory tract.

**Treatment.**—The following discussion applies primarily to the treatment of methyl alcohol poisoning. However, since certain other poisons may, on occasion, produce an acidosis of a similar nature, the treatment to be outlined is also recommended.

Special Measures in Industrial Cases.—The treatment of methyl alcohol poisoning seen in industry will differ to some extent from that seen in private practice. In industry, the toxic action will have usually been produced by absorption through the skin or by inhalation, in contradistinction to the ingestion of the product more commonly seen in private practice. For this reason the use of emetics is not indicated, and the value of gastric lavage with 4 per cent solutions of sodium bicarbonate will not be so great, although this latter procedure may be used and will at least be of value in treating the resulting acidosis.

Correction of Acidosis.—The most important phase of the treatment is directed at correcting the acidosis which is produced in the body by the formation of formic acid from the methyl alcohol. It has been the writer's experience to find that medical literature frequently advises treatment of acidosis but fails to give details of such treatment. For this reason, some detail in the treatment of acidosis is here included. In other sections where treatment of acidosis is suggested, reference to this description has been made. The use of sodium bicarbonate or sodium lactate intravenously is advisable. It is safest to calculate the dose necessary to restore the serum bicarbonate according to the method of Hartmann and Senn.

$$\text{mM.} = \frac{(60-CO_2)\ 0.7W}{2.24}$$

mM. = millimols of sodium bicarbonate or sodium lactate
$CO_2$ = serum carbon dioxide content in volumes per 100 cc.
W = body weight in kilograms

One millimol of sodium bicarbonate is 0.084 Gm.; 1 mM. of sodium lactate is contained in 1 cc. of r-molar sodium lactate. In marked acidosis when serum carbon dioxide is not known, 5 mM. of sodium bicarbonate (1.4 Gm.) per kilogram of body weight is a safe dose. Hartmann recommends the injection of one-half of the calculated dose of sodium lactate intravenously and one-half subcutaneously as one-sixth molar sodium lactate. R-molar sodium lactate is obtainable in sterilized ampules, which most physicians will find more convenient than sodium bicarbonate. *Sodium bicarbonate cannot be boiled or autoclaved in an unsealed vessel without forming the highly toxic sodium carbonate.* Sodium bicarbonate may be weighed and added with aseptic precautions to sterile water or to dextrose solution and injected intravenously in a 2 to 5 per cent solution. Both sodium bicarbonate and sodium lactate may be given by mouth when the patient is able to take them by this route.

Solutions for the Use in Acidosis.—Several very valuable solutions for use in treatment of acidosis are on the market. An interstitial salt solution (sodium chloride, 6.5 Gm.; sodium bicarbonate, 2.5 Gm.; and potassium chloride, 0.18 Gm. per liter) may be prepared by the addition of the contents of a 50 cc. ampule* to 500 cc. of sterile water. It may be used in practically all cases of acidosis without additional sodium bicarbonate and may be given subcutaneously or intravenously. Lactate-Ringer's solution (sodium chloride, 6 Gm.; sodium lactate, 2.7 Gm.; potassium chloride, 0.4 Gm.; and calcium chloride, 0.2 Gm. per liter) is equally valuable and is prepared in concentrated form in ampules,† the contents of which are to be diluted with distilled water.

Other Measures.—In addition to treatment of the acidosis the patient should be kept warm. Intravenous infusion of 10 per cent dextrose in physiologic saline may be of value in supporting him, and for this there has also been advised the intramuscular administration of atropine sulfate, 1/40 grain (0.0015 Gm.), not to be repeated; strychnine sulfate, 1/30 grain (0.002 Gm.); metrazol, 1½ grains (0.1 Gm.); camphor in oil, 1 to 2 cc.; coramine, 1.5 cc.; or caffeine sodium benzoate, 7½ grains (0.5 Gm.). Aromatic spirits of ammonia from ½ to 1 drachm (2 to 4 cc.) by mouth is said to be of value. If pain is present, the use of morphine sulfate, ¼ grain (0.015 Gm.), despite its central depressant effect, or codeine phosphate, 1 grain (0.065 Gm.), is frequently used; and if delirium is present, from 1/300 to 1/200 grain (0.002 to 0.003 Gm.) of scopolamine hydrobromide may be combined with the morphine.

Use of 50 per cent solutions of sucrose in treatment of cerebral edema if present has been suggested. The oral administration of a large dose of magnesium sulfate is usually part of the routine. With the exception of the eye, most abnormalities from chronic exposure clear up after removal from such exposure.

## The Glycols

The glycols result from the introduction of a second alcohol radical into the aliphatic hydrocarbons. Ethylene glycol possesses little narcotic properties but affects the kidneys; propylene glycol may be slightly irritant but has otherwise no effect; because of its high boiling point, diethylene glycol offers no industrial hazard.

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* Sharp and Dohme, Inc., Philadelphia, Pa.

† Eli Lilly and Co., Indianapolis, Ind.

Our chief interest is with the monoalkyl ethylene glycol ethers. The best known members of this group are ethylene glycol monoethyl ether, known to the trade as cellosolve, and the monomethyl compound, methyl cellosolve. The monoethyl compound is used extensively in spray painting, but no cases of industrial poisoning by this glycol have come to light. But with methyl cellosolve the case is different. The reason for this is probably that it is used in industry predominantly in processes requiring the application of heat to the substance, thus causing a high degree of volatilization. This danger has been observed in the shirt manufacturing industry, where a process known as "trubenizing" or "fusing" is utilized.

The first intensive investigation of methyl cellosolve was carried out by Greenburg[4] and associates of the Division of Industrial Hygiene, New York State. To that department had come reports that two brothers were suffering from "aplastic anemia, possibly due to benzol." In the plant under investigation they found that during winter, when the windows had to be kept closed most of the time, methyl cellosolve was present in the air at a rate of 76 p.p.m., together with ethyl alcohol, methyl alcohol, ethyl acetate, and petroleum naphtha. The workers were exposed to these substances in open vessels, open trays, and shallow pans near their pressing machines. The heat of the room and the heat of the pressing machines increased the volatilization of the solvents. A summary of the findings has been reported as follows:

"1. Clinical and hematological studies were made on 19 employees in the fusing department of a shirt factory where ethylene glycol monomethyl ether was used in conjunction with denatured alcohol as a solvent for cellulose acetate.

"2. Two workers who became acutely ill with a severe anemia were followed in the hospital and examined after discharge.

"3. One worker was examined who claimed that he had suffered from 'multiple neuritis' after working in the plant for three months the previous year. He was found to have an abnormal leukocyte picture a year after exposure had ceased, as well as symptoms and abnormal neurological findings characteristic of the group as a whole.

"4. The remaining 16 boys studied were at work at the time the examinations were made.

"5. All of the boys examined had abnormal blood pictures. Clinically, however, they fell into three main groups:

I. Those with a negative clinical picture.
II. Those with abnormal neurological findings on physical examinations and no symptoms.
III. Those with abnormal neurological findings on physical examinations and symptoms of fatigue and drowsiness.

"6. An abnormal blood picture, with or without neurological changes, may be present in the absence of symptoms. Reliance on a simple physical examination would thus be insufficient in these cases.

"7. Disturbances of erythropoiesis were found in nine cases.

"8. A general immaturity (shift to the left) of the neutrophilic granulocytes was found in every case.

"9. The blood platelets were fewer than normal in a majority of the cases and in several cases abnormal platelet forms were found.

"10. The reticulocytes, erythrocyte sedimentation rate, bleeding time, coagulation time, clot retraction, erythrocyte fragility and Van den Bergh tests were all within normal limits.

"11. The blood picture as a whole is believed to be suggestive of a macrocytic anemia associated with a reduction of the platelets and a relatively large number of young granulocytes.

"12. There appeared to be evidence of differences in the susceptibility of individuals here as elsewhere in industrial exposure.

"13. The responsibility of ethylene glycol monomethyl ether for these changes is suggested.

"14. It would seem desirable as a result of the present experience to keep the concentration of this substance in the air below 25 parts per million—an amount which it is believed may have been toxic in the cases reported."

Two subsequent laboratory investigations suggest that changes occur in the cellular elements of the blood and that consequently timely examination of the blood will serve to head off more serious results by demonstrating the early stages of poisoning. The differential diagnosis must rule out benzol from the offending substances.

### The Aldehydes

Aldehydes are oxidation products of primary alcohols. The group includes formaldehydes, acetalhyde, croton aldehyde, paraldehyde, and acrolein, of which the first and last named are of special importance from the point of view of industrial hazards.

**Occupational Exposures.**—Formaldehyde (HCHO) is used extensively in the form of a gas as well as in an aqueous solution (formalin) for disinfectant purposes in tanning, brewing, and glass etching, in the manufacture of inks and lacquers, and, more recently, in the production of plastics.

Formaldehyde is a marked irritant, but it also possesses weak narcotic properties. Inhalation of high concentrations may cause irritation to the lungs. The irritant action of this substance upon mucous membranes is due to its forming an irreversible combination with the protein of the surface cells. If absorbed in sufficient quantities, a toxic action occurs due to the breakdown of formaldehyde into methyl alcohol. However, because of its severe irritating action, inhalation of this gas is rarely extensive and therefore from the point of view of industrial health, its effect upon the skin must be regarded as of primary consideration. However, it acts not only on the skin, but also on the eyes, nose, and throat. In those exposed to amounts sufficient to be irritating, lachrimation is profuse and headaches sometimes present Those who are constantly exposed to small concentrations of formaldehyde may develop brownish discoloration of the finger tips, painful nailbeds, and fibrous areas.

**Prophylaxis of Formaldehyde Poisoning.**—For the prevention of the injurious effects resulting from exposure to formaldehyde, it is essential that its concentration in the air be kept below 10 p.p.m. and that contact of formaldehyde-containing dust or solutions with the skin be prevented by protective garments and proper personal hygiene. In operations in which there is a temporary massive exposure to dust, respirators should be worn, and the skin, especially that of the face, should be protected against the accumulation of dust. In case rooms or enclosures in which there is an appreciable concentration of formaldehyde gas have to be entered, workers should wear oxygen or open-air line masks. In judging the potential dangers of formaldehyde, it should also be kept in mind that with oxidizing agents formaldehyde might readily form carbon monoxide. For the prevention of more serious injuries to the skin, periodic medical examination of all workers handling formaldehyde or formaldehyde preparations appears to be indicated, and all persons showing signs of irritation of the skin and eczema should be excluded from further exposure until these conditions have completely healed.

**Treatment of Formaldehyde Poisoning.**—Formaldehyde solutions spilled on the skin should be removed immediately by washing with soap and plenty of water. In case of inhalation of formaldehyde vapors, the inhalation of vapors of diluted ammonia has been recommended.

Acrolein is an extremely irritating gas given off by the decomposition of fats under high degrees of heat. It is present in the synthetic rubber industry.

### The Ketones

Ketones result from the oxidation of the secondary alcohols. The lowest homologue of this series is acetone, $CH_3COCH_3$ which is used extensively as a solvent in industry. It has some mildly narcotic action, but no serious results from its industrial use have been reported in the literature. Acetone also possesses a degree of irritation to the mucous membranes of the eyes and of the respiratory tract.

Butanone, $C_2H_5COCH_3$, is a higher homologue of acetone and is capable of a more depressant action on the central nervous system. Butanone is also a greater irritant to the central nervous system than is acetone. Very few clinical studies have been made of the action of butanone. Smith and Mayers[5] had occasion to investigate exposure of workers to both acetone and butanone, without benefit of adequate exhaust ventilation, in the manufacture of raincoats. They report the following two case histories.

"CASE 1.—I. H., a girl brusher, 18 years of age, complained of gastric distress one morning in January while at work. Her tablemate also noticed that her eyes were watering. In the afternoon she felt worse and went to the rest room where she was found unconscious a short time later.

"An ambulance was called and she was taken to a nearby hospital about 4:30 P.M. She was admitted in coma with a strong odor of acetone on her breath. This caused the admitting physician to suspect diabetes, but the urine was negative. Physical examination was also negative except for occasional twitching of the face and extremities and hyperactive reflexes. Pulse 112. Temperature 98 degrees. B.P. 128/70. Spinal tap showed fluid under pressure—190 m.m. mg.

"She was given 6 cc. of coramin intravenously within an hour of admission and responded immediately. Her only complaint thereafter was severe headache. On the day after admission there were no objective signs. The headache subsided. She was discharged two days later.

"CASE 2.—M. M., likewise a girl brusher, aged 19 years, fainted while at work the day following the episode described above. The fainting was followed by a convulsion, after which she regained consciousness. She was taken to the same hospital where the admitting physician found her mentally confused and complaining of headache. No evidence was found in either history or physical examination of any pre-existing condition which might have been responsible for the convulsion. After an hour's observation, she was allowed to go home and two days later she returned to work."

Smith and Mayers imply that sufficient narcotic properties are present in these two substances to require the establishment of permissible limits and the maintenance of exhaust ventilation. It is also their opinion that while either acetone or butanone alone may be relatively harmless, that in combination they are able to provoke a synergistic effect.

### The Aliphatic Acids

These are ethyl formate, n-butyl formate, amyl formate, methyl acetate, ethyl acetate, and propyl acetate, to name the ones most frequently used in industry. The chief effect of exposure to the substances is an irritation to the eyes, nose, and throat. While it may be conceivable that under odd or compelling circumstances an individual might absorb sufficient amounts to suffer narcotic effects, the irritating properties of the aliphatic acids invariably prevent anyone from remaining in a contaminated atmosphere long enough for this to happen.

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# CHAPTER XI

# THE ALIPHATIC HYDROCARBONS—Cont'd

## THE ALIPHATIC OXIDES

### Carbon Monoxide

Carbon monoxide is the most important of the aliphatic oxides. Because it is one of the oldest of all poisons, the literature regarding the action of this gas has been prolific. Yet there still remains much confusion as to its effects, especially regarding the sequelae of acute carbon monoxide poisoning and the actuality of chronic poisoning, all of which demands clarification.

**Properties.**—Carbon monoxide is a colorless, tasteless, almost odorless, insidious gas with a density of 0.967 as compared with air. It can be compressed into a liquid and a solid. It burns with a blue flame. When combined with one volume of oxygen, it forms two volumes of carbon dioxide. Carbon monoxide originates from combustion of carbonaceous material.

**Occupational Hazards.**—Because it is practically ubiquitous, it is impossible to list every hazard. The more common sources are electric furnaces, blast furnaces, gas manufacturing plants, oil distilleries, charcoal ovens, refuse plants, kilns, and coal mines. To a lesser extent for acute poisoning, but a common source for chronic exposure, is the occupation of presser in tailor shops, laundry workers, garage mechanics, furnace tenders, firemen, cooks, and housewives. Broadly, anyone who works where power or heat is being generated by a combustion process of carbonaceous material may be exposed to carbon monoxide poisoning.

**Pathology.**—As a prelude to the discussion of the pathology of carbon monoxide it would seem appropriate to mention certain physiologic phenomena of this gas.

*Types of Asphyxia.*—CO belongs to the group of gases classified as asphyxiants. Of these there are two divisions: (1) Those which result in asphyxia due to a cessation of breathing. This may be from a mechanical obstruction to normal respiration or to the action of drugs upon the respiratory center. Strangulation, drowning, or confinement in an oxygenless vault or cave-in of dirt are examples of mechanical asphyxia. Morphine and chloroform, acting upon the respiratory center, are examples of the action of drugs. (2) Those which result in asphyxia from a deficiency of oxygen, but where breathing is not interfered with.

*Comparison of Cyanide and Carbon Monoxide Poisoning.*—This second type of asphyxia is produced by two chemicals, carbon monoxide and cyanide, each of which acts differently within the body. As pointed out under Cyanides, the cyanides poison the tissue cells in all parts of the body to prevent the entrance of oxygen into the cells. Carbon monoxide, on the other hand, reacts within the blood stream. While carbon monoxide enters the body only through the lungs, it causes no immediate reaction within those organs. During acute carbon monoxide poisoning there exists no interference with oxygen entering the lungs in sufficient quantities. But after the initial reaction with the CO, the blood can no longer absorb what oxygen the lungs supply. In cyanide poisoning the oxygen enters the body and is transported by the blood stream, but the gate is closed against its entering the various tissue cells of the body.

*Supplanting of Oxygen in Blood Stream by Carbon Monoxide.*—As carbon monoxide enters the blood stream, it immediately combines with the hemoglobin and replaces the oxygen. The normal oxyhemoglobin is therefore converted into carbon monoxide hemoglobin, as illustrated by the following equation: $HbO_2:CO::HbCO:O_2$. This reaction is reversible, for as oxygen is supplied and carbon monoxide removed, the oxygen supplants the carbon monoxide existing in the blood stream in combination with Hb to form again oxyhemoglobin. It should be noted that the red cells which carry this carbon monoxide are not injured in the process. Finally, concerning this interchange of gases, it is important to know that carbon monoxide has a great affinity for hemoglobin, more so than oxygen, this affinity being estimated at approximately 300 to 1.

GENERAL CHANGES.—The body at the time of death may show cherry-red blotching in various areas. Upon opening the body, one finds the blood bright red. It is usually coagulated but at times fluid. Hemorrhages may be present in the lungs, pleura, pericardium, intestinal tract, and uterus and under the mucous membranes of the body. Regarding the heart, there is a marked lack of sufficient autopsy reports to enable investigators to formulate a definite conclusion concerning the cardial damage which may result from CO poisoning. To date the evidence would indicate that there is no marked damage to the heart or coronary vessels unless the exposure has been of the severest type. The lungs usually reveal no special change unless the period of unconsciousness has been prolonged.

CHANGES IN CENTRAL NERVOUS SYSTEM.—Primarily, the central nervous system sustains the greatest injury as a result of carbon monoxide poisoning, although the nerve tissue of any part of the body may be involved. Vascular lesions constitute a prominent part of the pathologic alteration, the arteries as well as the veins being involved. In the brain the vascular stasis is pronounced, the vessels being dilated and packed with red blood cells. Petechial hemorrhages are often numerous and scattered. Following the vascular congestion, edema ensues. The increased congestion and accumulative edema will cause a rise in the intracranial pressure. Because of this widespread involvement of the brain, a victim who survives a sublethal but severe exposure may show evidence of disseminated encephalitis, bilateral softening of the lenticular nucleus, internal hydrocephalus, or a combination of these lesions.

**Diagnosis.**—

SIGNS AND SYMPTOMS.—Following the work of Haldane, Henderson, and Haggard and Sayers and Yant, of the Public Health Service of the United States and the Bureau of Mines, Hamilton[1] formulated the data given in Table IV.

TABLE IV

| PERCENTAGE OF CO IN AIR | EFFECTS |
|---|---|
| 0.02 | Possible headache, mild frontal, in two to three hours |
| 0.04 | Headache, frontal, and nausea after one to two hours<br>Occipital after two and one-half to three and one-half hours |
| 0.08 | Headache, dizziness, and nausea in three-quarters of an hour; collapse and possible unconsciousness in two hours |
| 0.16 | Headache, dizziness, and nausea in twenty minutes; collapse; unconsciousness, and possibly death in two hours |
| 0.32 | Headache and dizziness in five to ten minutes; unconsciousness and danger of death in thirty minutes |
| 0.64 | Headache and dizziness in one to two minutes; unconsciousness and danger of death in ten to fifteen minutes |
| 1.28 | Immediate effect; unconsciousness and danger of death in one to three minutes |

The signs and symptoms of carbon monoxide poisoning fall into one of three groups: (1) acute asphyxiation, (2) acute asphyxiation with delayed symptoms, and (3) chronic poisoning.

ACUTE ASPHYXIATION.—As shown by Table IV, this may vary in its extent, being dependent upon the concentration of the gas, the activity or inactivity of the patient, and the individual susceptibility. Headache and dizziness may be noticed with possible nausea if the concentration does not immediately overcome the patient. Survivors have related a throbbing type of headache with roaring in the ears, confusion, and general weakness preceding unconsciousness. Unconsciousness occurs when nearly half the hemoglobin is bound to the CO. If bodily effort is made during the exposure, unconsciousness comes on more quickly. It is thought that CO is eliminated in from twelve to twenty-four hours.

Patients usually recover consciousness at a 50 per cent CO-hemoglobin level if the poisoning occurred suddenly and the patient is not long in the contaminated atmosphere. The duration of exposure is more important than the percentage of CO in the determination of severity and in the production of permanent sequelae.

ACUTE ASPHYXIATION WITH DELAYED SYMPTOMS.—Symptoms and neurologic changes may be noticed immediately, or there may be a delay of from a few days to several weeks. Based upon the recognized pathology, most authorities contend that signs or symptoms must appear within the first three or four weeks. After that time they are to be considered as coincidental and unrelated. This contention is frequently questioned in medicolegal cases, as seen in Case I. In the unquestioned case, the picture is varied. Headaches and dizziness may ensue and persist. Visual deficiency or blindness may be present. Twitching. choreiform movements, or convulsive seizures occur. The emotional life and will power may be profoundly altered. Apathy, disinterest, dulled memory, lack of judgment, and in fact, the whole gamut of mental changes even to idiocy or imbecility has been noted. There may be confusion, hallucinations, states of excitement, and symptoms suggesting general paresis. Parkinsonism is not unusual.

In addition to the effect upon the central nervous system, pneumonia may set in within a few days following the acute exposure. Deglutition pneumonia following vomiting of food has been reported. There are also references in the literature to complications following hemorrhage into the kidneys, spleen, or liver. Permanent damage to the heart is highly controversial.

CHRONIC CARBON MONOXIDE POISONING.—The expression "chronic carbon monoxide poisoning" is a misnomer but has been used as an introduction to this portion of the chapter for want of a better term. Carbon monoxide is not cumulative. The regulation of working hours is such that within twenty-four hours workers live in an oxygen-pure atmosphere at least twice as long as in contaminated air. Exposure to small amounts is therefore not likely to produce any pathologic changes. Most of the subjective symptoms attributed to carbon monoxide may well be the result of nonindustrial disease, which, upon examination, they usually prove to be. Too seldom in cases of this type is sufficient cognizance given to conditions of substandard living, malnutrition, and the like.

In several articles Beck and associates[2,3] have claimed that a wide variety of pathologic changes is due to protracted exposure to minute or small amounts of carbon monoxide. Some of the diseases they refer to concern the nervous system, the cardiovascular system, and the gastrointestinal system. The list of diseases which these authors attribute to an extended exposure is surprisingly long, and many authorities in the field have voiced their disagreement. In a previous textbook[4] this author devoted considerable space to a refutation of Beck and associates to which the reader is referred, since it seems to be more instructive to proceed at this time by quoting two other satisfactory authorities. It was unfortunately too late to include a reference to an excellent article by Yandell Henderson[5] in the volume on Occupational Diseases. That author was greatly disturbed over Beck's articles, and stated:

> "Whether there really is such a disease as that termed 'chronic carbon monoxide poisoning,' as a consequence of prolonged or repeated exposure to small amounts of the monoxide and continuing after the exposure is stopped, is very doubtful. Certainly its importance in many cases is less medical than legal and financial as a basis for claims for damages to be awarded by a compensation commissioner or a court of law. There is a large literature presenting numerous cases, described with clinical fullness and accuracy in respect to the symptoms of the disorder but with little or no evidence that the patients had ever been exposed to any significant extent to carbon monoxide.
>
> "If, then, numerous false claims are not to be bolstered by such papers, it is essential that writers on this subject should be required to report evidence as to the analytic method by which the amount of carbon monoxide in the air or in the blood was determined, how much was found and how long the exposure continued. Such a requirement would at once deprive the greater part of the literature in this field of all evidential value.
>
> "The question remains whether repeated exposures to amounts of carbon monoxide only sufficient to induce a headache but falling considerably short of rendering a man unable to stand or drive a car can do more than impair health temporarily. At present the probability is that termination of the unhealthful condition is always rapidly followed by return to health. Certainly the men who drive taxis in our large cities and those who work in repair shops, although liable to all the disorders mentioned by Dr. Beck and his co-authors, have not yet been shown to be appreciably more liable than the rest of the population. We all breathe some carbon monoxide nearly every day of our lives.
>
> "The expression 'chronic poisoning' can be properly applied only to the action of substances that are protoplasmic poisons and that may accumulate in the body. Lead is a typical example. Carbon monoxide, on the contrary, is rapidly eliminated by respiration, and its action while in the blood is wholly due to the temporary displacement of oxygen. The anoxia due to carbon monoxide acts essentially as does that induced by the low pressure of the air (i.e. oxygen deficiency) at great altitudes. The headache of mountain sickness and that of a temporary 20 or 30 per cent saturation of the blood with carbon monoxide are identical. Yet, given time for compensatory physiologic adjustments—of which an increase of red corpuscles in the blood is one—men are quite healthy at Denver and even on Pike's Peak."

To Henderson's valuable opinion can be added that of Rossiter,[6] who wrote in *Industrial Medicine:*

> "By chronic carbon monoxide poisoning is meant, the alleged effects produced by the inhalation of low concentrations of this gas over a long period of time, and should not be confused with the true after-effects sometimes seen following severe gassing where the victim has been unconscious for a long time. After a careful study and an extensive investigation, together with my personal observations over a period of 34 years, during which time over 3000 cases were

treated, I do not believe that such a condition as chronic poisoning exists. No definite proof or convincing evidence has been given, and the leading authorities on the subject have not accepted it as a fact. Practically everyone admits the gas is non-toxic and that the effects are due to oxygen-want, but those who talk about chronic poisoning fail to take into consideration that there is no anoxia unless there is severe gassing and that the victim must be rendered unconscious. It is true that men exposed to small quantities of gas over a period of time attribute various complaints to the gas, but I have arrived at the conclusion that all their symptoms can be traced to some underlying condition; such as, errors in diet, unhygienic living, alcoholism, syphilis, bad teeth and tonsils, rheumatism, etc., which when corrected gives immediate relief. Such symptoms complained of as headache, dizziness, palpitation, digestive disturbances, nausea, tired-out feeling, loss of memory, anemia, insomnia, general weakness, loss of sexual desire, constipation, etc., may result from many different causes. These symptoms cannot be distinguished from the symptoms of ill health which are generally associated with age and the nature of one's work. In making a diagnosis as to the cause of these symptoms, it is necessary to take into consideration many factors pertaining to work, life and general health. Work may compel a man to stand or sit all day; he may be subjected to excessive heat and humidity; the ventilation and general working conditions may be poor; he may be forced to work under artificial light; there may be extreme noise and vibration about him, and dust and other fumes may play some part. He may have marital or financial worries. Poor food, errors in diet, dissipation, unhygienic living are great factors, as well as bad teeth, bad tonsils, gout, rheumatism, exposure, syphilis, and alcoholism. When we consider all these things and our present knowledge of the mode of action of this gas on the human system, I believe the possibility of carbon monoxide causing any chronic condition is very remote. However, it must be remembered that contaminants in the gas may cause ill-effects."

Truck drivers who make long hauls are subject to exposure which might be termed subacute. In a survey conducted by the California State Industrial Hygiene Service in conjunction with the State Highway Patrol, 1,105 commercial motor vehicles of various types were tested in five-minute runs on the highway under varying weather and road conditions. It was found that 2.9 per cent of these vehicles were in a potentially dangerous condition due to the fact that in the driver's cab carbon monoxide in a concentration of over 0.01 per cent could be ascertained. This amount, if inhaled for a period of six hours, is sufficient to cause headache, drowsiness, faulty judgment, and impaired driving ability. A different type of traffic problem occurred at the U. S. Immigration Service gates at the California-Mexican border, where an attendant was overcome by carbon monoxide fumes. During the heavy traffic hours the concentration of these noxious exhaust gases reached two to three times the limit of safety.

TESTS.—Whenever possible, all individuals with suspected carbon monoxide poisoning should be subjected to accurate quantitative tests. Unfortunately, the average physician cannot carry out the elaborate procedures entailed in such an analysis. For more definite information as to the methods of quantitative analysis, reference to the literature is made at the end of this chapter. For those occasions when adequate laboratory procedure is not available but when confirmatory evidence of the presence of carbon monoxide in the blood is desired, the following tests are suggested:

*Tannic Acid Test of the Blood.*—Sayers and Yant's tannic acid method gives an approximative quantitative estimate of the CO present. In a small test tube dilute 0.1 cc. of suspected blood with 0.4 cc. of dilute ammonia (1 cc. of strong ammonia per liter). Mix and add 1.5 cc. of freshly prepared 1 per cent tannic acid solution. Let stand overnight and compare the color either with permanent standards or with standards prepared simultaneously as follows: Bubble CO or illuminating gas through 1 cc. of oxalated blood until saturated. Dilute this and also 1 cc. of normal blood with 4 cc. of dilute ammonia. Make up a series of mixtures of these laked bloods (in 0.5 cc. volume) corresponding to 10, 20, 30, 40, 50, and 60 per cent saturation. Add 1.5 cc. of tannic acid solution to each.

In no test are the color differences well marked if the CO saturation is under 20 per cent. In acute poisoning, the saturation usually ranges from 25 to 60 per cent or more, but it falls quickly after the patient is removed from the source of poisoning (30 to 50 per cent per hour in air, about four times as fast in 95 per cent O and 5 per cent $CO_2$).

Another simple test is merely that of boiling some withdrawn blood. Ordinary blood becomes brown-black, while blood rich in CO becomes a brick-red mass. While easy to do, this test is devoid of any estimate of percentages.

A handy, quick determination is that of taking 2 cc. of carbon monoxide blood with an equal quantity of water and adding three drops of one-third saturated solution of copper sulfate. This will give a brick-red precipitate, while normal blood will yield a greenish-brown one (Zaleski).

**Differential Diagnosis.**—An authentic history of exposure, the absence of the odor of alcohol on the breath, and the presence of the symptoms previously outlined will indicate a CO poisoning. Difficulty lies in the variation of degree of intoxication resulting in a simulation of other diseases. Drug poisoning, alcoholism, cerebral and cardiac accident, and coma from diabetes or uremia must be considered. Suicidal intent must be differentiated from industrial exposure. Finally, *a diagnosis of acute carbon monoxide poisoning is never positive unless determined by an examination of the blood.* In instances where carbon monoxide is suspected but where it has disappeared from the blood stream, it may be found hours later in the spinal fluid.

**Sequelae of Acute Carbon Monoxide Poisoning.—**

1. When the dose has been sublethal and the exposure short, recovery rapidly takes place within a few hours or a day or two at the most, without residual symptoms or signs.

2. If the dose has been sublethal but severe, (a) the patient may recover within a few days to continue his previous state of health without ever showing any subsequent evidence of carbon monoxide poisoning or (b) signs or symptoms may be noted upon the patient's emerging from the initial coma; these may persist for several days, several months, or for the duration of the victim's life; or (c) following the unconscious period, the victim may be apparently normal for a few days to several weeks, only subsequently to reveal nervous or mental impairment. Here again the length of the impairment is indefinite. It may be temporary, lasting for several weeks to several months, or may be a permanent change.

EFFECT ON LIVER, SPLEEN, KIDNEYS, AND LUNGS.—There is no volume of evidence that severe acute exposure affects these organs in any permanent manner. The lungs will invariably reveal moisture immediately following the gassing. This will soon disappear, or pneumonia may be a complication, although this is not common. However, pneumonia, to be attributed to exposure to this gas, should ensue within a few days to a week following the exposure. Tuberculosis is not a sequela. Glycosuria is frequently present in acute poisoning from this gas. It usually disappears within a few days, and normal liver function is resumed. It is held that the mobilization of sugar in such instances results from central nervous system impairment during the phase of asphyxia. Some differ from this view, placing the responsibility upon the adrenals or the thyroid, or both. While the urine also reveals albumin, this likewise soon clears up and rarely is there permanent damage to the kidneys.

The previous paragraph indicates that the majority of the systems of the body escape damage to any appreciable extent from an assault of carbon monoxide. Two systems, not mentioned in that paragraph, deserve more detailed consideration; namely, the nervous and circulatory systems.

EFFECT ON NERVOUS SYSTEM.—From what has been written, it is obvious that the central nervous system exceeds all the others in its frequency of injury. Yet the frequency of permanent damage does not parallel to any extent the incidence of injury. The profession and laity have an erroneous belief that patients who show early mental or nervous changes will continue to do so for the rest of their lives. Probably the best concrete rebuttal to such a belief is to be found in the report by Schillito and associates.[7]

In the New York City area from 1925 to 1935, there were more than 21,000 cases of acute intoxication from carbon monoxide. Schillito and co-workers reviewed these records, as well as the records of seven state institutions that receive the mental and neurologic patients from that city. From this investigation they estimated that only one in every

500 patients with acute carbon monoxide poisoning later showed mental or nervous symptoms. Regarding subsequent psychoses, he found only one case of carbon monoxide psychosis to 2,000 other types of psychoses, or a percentage of 0.05.

Effect on Heart.—Does acute carbon monoxide poisoning produce cardiac changes of a permanent nature? Concerning the occurrence of nervous system lesions due to CO intoxication there exists no lack of evidence, but regarding permanent heart lesions, dogmatic opinion should be withheld until reasonable scientific deductions can be made from a large series of autopsied cases, and also from a series of cases in which the state of health was known before exposure and in which these patients have been followed for a long period of time after exposure. The consensus in the literature is that, aside from the expected imposition placed upon the heart at the time of gassing, there persists no cardiac damage. Invariably, autopsy reports describe profusely the pathologic changes in the central nervous system but only occasionally refer to findings in the heart, which would suggest a coincidental relationship rather than a consistent sequela. White,[8] in discussing remote causes of heart changes, says:

"Some changes in the heart have been reported, as for example, subendocardial hemorrhage after CO poisoning, but such changes have been neither extensive nor consistent. Chronic symptoms and signs, such as the inverted T waves in the electrocardiogram resembling coronary T waves, should be ascribed to pre-existing heart disease, whether or not accentuated or revealed after CO poisoning, about which especially there has been so much uncertainty."

*(a) Electrocardiographic Changes.*—Stearns, Drinker, and Shaughnessy[9] produced an electrocardiographic study of a series of cases of carbon monoxide asphyxia, to find that the most common deviation is an abnormality in the T waves or in the level of the S-T segment. They feel that the changes, having been *transitory*, may be ascribed to carbon monoxide asphyxia, except when coronary disease may have been a contributing cause in some cases.

*(b) Basis for Diagnosis of Cardiac Injury.*—There is no intention in the previous statements to imply that permanent cardiac damage cannot occur following a severe, prolonged exposure. In such instances signs and symptoms referable to the heart should be in evidence immediately or soon after the exposure. The condition should be followed by periodic examination; and if it is apparent that the impairment is of a permanent nature, the workman should be accorded compensation. But the onset of a heart lesion months or years after an exposure to carbon monoxide poisoning deserves no consideration as to their relationship.

**Treatment.—**

PROPHYLAXIS.—The prevention of carbon monoxide poisoning in industry is not ordinarily a difficult problem. In the larger industries where combustion of various types is a part of the process, the hazard is well recognized and, therefore, precautions are taken. Small plants and garages constitute a more likely source of this poisoning. Adequate ventilation is all that is necessary to insure against poisoning from this gas, but men need to be warned about closing windows and doors, which they are prone to do in the winter months.

ACUTE ANOXEMIA.—The victim should immediately be removed from his causative environment and taken into the open air where artificial respiration should be maintained until oxygen therapy can be instituted. For the ideal treatment, oxygen should be applied under pressure of three atmospheres (30 pound gauge). At this point, medication of any type is not only ineffectual but actually harmful. After the patient has been resuscitated, preventive measures against the development of pneumonia are indicated.

CASE 1.—A 59-year-old night mechanic was found unconscious in a garage near the running motor of a truck. He was apparently normal two hours previously when he dispatched a truck and driver from the place. After preliminary attempt at resuscitation he was taken to a Long Beach hospital, where a diagnosis of carbon monoxide poisoning was made. After remaining in the hospital for one week, he went home but continued to report thrice weekly to his physician for treatment to a leg burn suffered at the time of the attack. After seven weeks he returned to work and continued at that for two months, at which time he was discharged by his employer.

Following his discharge, his wife filed claim for permanent total disability, stating that her husband had undergone a change in personality as a result of the carbon monoxide poisoning. This change, she alleged, consisted of loss of memory, apathy, and disinterest, inability to concentrate sufficiently to finish any project he attempted, and other conditions. Six months after the alleged poisoning and just prior to the trial, the patient was referred to the writer.

*History.*—The past medical history was irrelevant. With the exception of poor oral hygiene and slight bilateral edema of the ankles, the general examination was negative. The heart action and cardiac history did not account for the edema. The neurologic examination was entirely negative. The patient had a solid, quiet, reticent manner, but his expression did not impress the examiner as being typical of the masklike countenance of parkinsonism. He readily answered all questions, was well oriented as to time, place, and events and smiled when humorous remarks were made. The laboratory investigation was entirely normal.

*Testimony.*—The following facts were brought out at the trial. Sometime before he lost consciousness (the patient does not know how long), he had a severe pain around the heart while outside the garage. When he was taken to the hospital *no blood test was made* for the presence of CO. The wife stated that she noticed definite mental changes in her husband two or three days after the accident. The physician who first cared for him had recorded no such changes in the progress notes or in his office record. The employer testified that he had noted changes in the character of the patient's work prior to his accident. The psychiatrist who testified for the claimant believed the man had parkinsonism as a result of carbon monoxide exposure. A second psychiatrist who examined the patient, but who did not testify, reported by letter that he believed the man did not have parkinsonism and was able to work. The author testified that while the circumstantial evidence strongly indicated a carbon monoxide poisoning, this diagnosis could not be established in the absence of any blood test, and such a diagnosis, while likely, was clouded by the patient's statement that some hours previously he had had a severe heart pain. Furthermore, the author felt that it was impossible for him to evaluate any alleged change in this man since he did not observe him prior to this accident, and other than the wife's statement, there was no evidence that such a change had occurred. Certainly to him the case was not a definite parkinsonism.

The commission found that no evidence had been produced to show that the claimant was either mentally or physically unfit to work as a mechanic.

The case is presented not because it adds anything to the knowledge of carbon monoxide poisoning but because of certain lessons which can be drawn from it. These lessons are to be found in errors of omission committed by the physician in attendance. Had the patient been overcome in his own home, these omissions would not have been so significant. First, while the circumstances strongly indicated a carbon monoxide poisoning, the diagnosis was never established because of the failure to examine the blood. It subsequently developed that a possibility of a cardiac episode clouded the issue. Second, the wife's contention that mental changes set in shortly after the accident could not be denied or substantiated by the physician's hospital record or office file. He failed to appreciate the possibility of the development of latent effects. It should be apparent to the reader, then, that a case of carbon monoxide poisoning arising out of occupation should first be established by blood investigation for CO and then observations for sequelae recorded over a period of time.

CASE 2.—A presser in a tailoring establishment claimed loss of weight, fatigue, and two attacks of coma due to chronic exposure to gas fumes. His physician, upon the basis of history, diagnosed the case as chronic carbon monoxide poisoning. He was given from three to six months' temporary disability and advised not to return to work. The insurance carrier desiring to determine the authenticity of the claim was unable to locate him for three months, but at the end of this time found him visiting relatives in Arizona. Investigation revealed that his mother had diabetes; he had been refused insurance because of sugar in the urine eight years previously; he had polyuria, polydipsia, excessive appetite, glycosuria, and a blood sugar of 280 milligrams. Investigation of his working conditions revealed that he pressed but rarely, usually being engaged in repairing and mending. The machine he did operate when pressing was located by an open window.

The case was not compensable. Preponderant evidence pointed to diabetes mellitus, and the occupational conditions indicated the improbability of exposure.

CASE 3.—An unmarried professional man, aged 27 years, was found unconscious with a companion in a parked automobile on the morning of Feb. 18, 1934. The estimated duration of exposure to carbon monoxide was about six hours, and another three hours elapsed before he was brought to the hospital. The companion was dead when found, which indicated the severity of the exposure. The windows of the car were closed, the engine had been left running, and later investigation revealed a leak in the gas heater with which the car was equipped.

On admission to the hospital, physical examination showed the patient to be in profound coma; he was cold and clammy; there was extreme cyanosis of the face and extremities; and the lips were cherry-red. The heart tones were of good quality. The lungs showed moist râles, and breathing was greatly labored. The abdomen was distended and tympanitic. There had been a profuse spontaneous diuresis before the patient was moved from the car. The pupils were widely dilated and reacted only slightly to light; corneal reflexes were absent. The only other neurologic feature of import was a bilateral Babinski sign.

For the first thirty-six hours after his admission to the hospital the patient remained in a profound coma. For the next thirty-six hours the coma was apparently less profound, and the patient began to show some involuntary movements, moving his head and hands aimlessly. At this time he was also able to take fluids orally. On the following day a more noticeable restlessness developed; the patient opened and closed his eyes, snapped his fingers, and thrashed about in his bed. This behavior continued for nearly two weeks with but little change. Sixteen days after admission the patient rather abruptly regained consciousness and recovered his power of speech. Neuropsychiatric examination at this time showed a marked clouding of the sensorium.

Attempts to converse with him elicited only brief, but not entirely pointless, remarks. Asked, "What is your name?" he replied, "Puttentain", a response suggestive of a childhood game which the patient had known. To other questions relative to his background he gave abusive retorts as, "Who the hell wants to know?" He showed no evidence of recognizing his mother or sister, with whom he had been living at the time of the accident, or other close relatives or friends. Questions relative to his physical condition brought a more adequate response, and he complained of pains in the legs and feet.

During the days immediately following he was overtalkative and showed a marked flight of ideas. He was irritable, profane, obscene, combative, argumentative, and extremely uncooperative. He showed an almost complete loss of memory for recent and remote events. He demonstrated some of the components of the parkinsonian syndrome in a marked degree. These consisted of pill-rolling, drooling from the mouth, and, to a lesser extent, the masklike facies. This condition was evident on the sixteenth day after exposure to carbon monoxide and persisted for over a month, disappearing gradually.

After he had regained consciousness the patient suffered severe pains in the arms and legs and was very voluble in his complaints about this condition. The toe drop on the right was noticed on examination of the limbs. The peripheral neuritis responded rather slowly to baking and massage, and except for mild paralysis of the muscles of the right foot, which was aided by a support, the patient made a complete recovery. He became oriented in all spheres, regained his memory for remote and recent past events, and returned to his normal mental level by the sixtieth day after admission. He was discharged seventy-seven days after admission, but he was unaware of the nature of his illness before this time. On being informed of the circumstances of the accident resulting in his illness, he showed a normal concern for his dead companion and for the possible consequences to himself and his relatives.

About a year after the accident the patient was able to resume his professional activities in a limited way, and a year after this, (two years after the accident) he had so far recovered that he discharged his full duties without any noticeable impairment.

This case illustrates the severity of exposure, the onset of neurologic signs and symptoms within the expected two- or three-week period, and the eventual recovery after a temporary disability of two years.

### Carbon Dioxide, $CO_2$

Carbon dioxide is a colorless, odorless, heavy gas. It is the product of respiration, of combustion from coal and wood, of decay, and fermentation. It may be found in the environment of blast furnaces, large refrigeration plants, boiler rooms, breweries, large fermentation vats, submarines, and the holds of ships.

The signs and symptoms of carbon dioxide absorption are excitement, headache, vertigo, dyspnea, drowsiness, weakness, dizziness, and muscular weakness. Following initial excitement, high concentrations may result in coma or even death. In solid form, as carbon dioxide snow, also called dry ice, burns of the skin will result if protective measures are ignored.

Temporary disability from exposure is always of short duration, and no permanent disability results. A case in point is that of a longshoreman entering the hold of a ship to unload fruit from South America which was in a marked state of decay. He was overcome, and two workers who rescued him were also partially overcome. All victims recovered completely within twenty-four hours.

### Carbon Oxychloride, $COCl_2$

This is more commonly known as phosgene. It is a very irritating gas which affects all mucous membranes but is especially harmful to the pulmonary tissue. Phosgene may result from the decomposition of certain chlorinated hydrocarbons when these substances are in contact with the naked flame.

## ALIPHATIC NITRILE COMPOUNDS

Of these compounds, cyanogen (NCCN), hydrocyanic acid (HCN), and cyanogen chloride (CHCl) are the most important. These substances have a wide use in the extraction of gold and silver from their ores, in case hardening, in electroplating, in fumigation, in the spraying of trees or orchards, in photography, in pickling, and in other industrial processes.

**Pathology and Physiology.**—As stated in the discussion of carbon monoxide, the cyanides are protoplasmic poisons, exerting an inhibiting action upon tissue oxidation. Unlike carbon monoxide, the cyanides do not combine with hemoglobin. It should be understood that cyanides do not block oxygen from reaching the blood, or from being transported by the blood; but once the cyanides reach the tissues, they inhibit there the use of oxygen. On the other hand, hydrocyanic acid combines readily with methemoglobin to form cyanmethemoglobin, a fact which has been taken into consideration in the newer methods of prophylaxis and therapy. Cyanogen as well as hydrocyanic acid has the ability to arrest oxidation in the tissues. In addition to this quality, cyanogen chloride or cyanogen bromide acts as a strong irritant to the pulmonary fields.

There result various pathologic changes. If the body is opened shortly after death, a sweetish odor which some have described as a "peach kernel odor" will be observed. The blood is bright red and uncoagulated, and the veins as well as the right heart appear congested. If the patient has lived for several hours after onset of poisoning, there is likely to be generalized engorgement of the lungs, liver, kidneys, and spleen. Petechial hemorrhages are not uncommon. However, in the majority of cases where death has occurred rapidly, little or no changes will be noted.

Most laymen and indeed many physicians assign the same toxicity to the cyanides as to hydrocyanic acid; yet the former are only about one-fourth as dangerous and are used extensively in industry with but rare instances of harm. This almost universally safe technique in the handling of the cyanides is based on the prevention of any action which would liberate hydrocyanic acid.

**Signs and Symptoms.**—Poisoning may occur by absorption through the skin, cuts, or abrasions, by ingestion, and by inhalation. If the poisoning is not immediately fatal or if the dosage is just sublethal, there is constriction of the throat, headache, and vertigo; respirations are rapid but grow progressively slower if the intoxication becomes more profound. As coma ensues the eyes become glassy, convulsive twitching may be present, and often the patient will foam at the mouth. Milder forms of acute intoxication will result in headache, vertigo, and nausea.

Whether or not there exists a chronic form of cyanide poisoning is a debatable question. In workers exposed daily to such processes as electroplating or pickling, a cyanide rash characterized by itching and by macular, papular, or vesicular eruptions may occur. Smith[10] reports several cases which she attributes to chronic cyanide poisoning. In one of these cases a man, three years after his removal from exposure, exhibited wasting of the muscles and a disabling paralysis. It is difficult for the writer to believe that these symptoms could justifiably be attributed to cyanide, in view of its short reaction within the tissues as well as the fact that the average workman is living in an atmosphere free from this substance for at least twice the length of time which he is likely to spend under its influence. It is conceivable that cyanide, like carbon monoxide, could produce general malaise, but it does not seem plausible that it should be able to cause paralysis or other disabilities of a permanent nature.

**Tests.**—Reagents are:

1. Ferrous sulfate, 1.5 per cent; ferric chloride, hydrochloric acid, and sodium hydroxide, each 10 per cent. To about 10 cc. of the suspected fluid (blood or gastric contents, etc.) two drops of the sodium hydroxide are added. Then, following the addition of 2 cc. of ferrous sulfate and 1 cc. of ferric chloride, the mixture is warmed, and hydrochloric acid is cautiously added, avoiding any excess. A blue precipitate shows the presence of hydrocyanic acid.

2. Dissolve 10 mg. of ferrous ammonia sulfate and 10 cc. of ammonium acetate (5 to 10 per cent) in 50 cc. of water. If 1 cc. of this solution is added to a small amount of blood containing cyanide, a purplish precipitate occurs.

3. Kobert's examination of the blood using the spectroscope: Two small bottles are filled with normal and suspected blood diluted 1 to 100. Both of these exhibit the absorption bands of oxyhemoglobin. The two bottles are corked and placed in a warm dark place for twenty-four hours. During this period, the normal blood has become darker in color and will show absorption bands of reduced hemoglobin. If the suspected blood also darkens and gives absorption bands of reduced hemoglobin, no cyanide was originally present, but if the suspected blood remains bright red and still gives the absorption bands of oxyhemoglobin, then cyanide was originally present.

4. If a piece of starch paper, which has been made slightly blue with a trace of iodine, is placed in the suspected solution, it will be decolorized at once if even a trace of cyanide is present. Dilute to 1 part in 1,000,000. Although this test is extremely delicate, the reaction is obtained by hydrogen sulfide, sulfur dioxide, uric acid, albumin, and alkalis.

5. To 5 cc. of distillate add a few drops of potassium nitrate solution, then two to four drops of ferric chloride and a few drops of sulfuric acid to get a bright yellow color. Heat to boiling. Add sufficient sodium hydroxide solution to remove the excess of iron and filter. To the filtrate add two drops of dilute ammonium sulfide solution. If the solution contained hydrocyanic acid, a violet color will appear and pass through blue, green, and yellow. This test is delicate to 1 part in 312,000.

**Medicolegal Aspects.**—Temporary disability: After surviving an acute poisoning, the patient invariably recovers within a few hours to a few days. Those individuals alleging symptoms from chronic exposure can be expected to recover within several weeks after removal from exposure. Permanent sequelae are not to be expected.

**Treatment.**—The use of a solution of methylene blue has been discredited. If the patient has ingested a cyanide, which might conceivably happen in industry but seldom actually does, alternate intravenous injections of sodium nitrite and sodium thiosulfate are recommended. If poisoning has occurred by inhalation, the most rapidly effective antidote is the immediate inhalation of amyl nitrite for thirty seconds every two minutes.

SODIUM NITRITE TREATMENT.—Give at once a slow and careful intravenous injection of 1 per cent sodium nitrite solution, in five divided injections, until 50 cc. are injected in about one hour. If improvement is manifested, but prognosis is still unfavorable, the injection may be cautiously continued but is to be stopped at once in case of sudden collapse. Epinephrine, 1 cc. of 1:1000 solution, should be ready at hand to combat nitrite shock, if necessary. Fortify the nitrite treatment at once with the intravenous injection of 20 cc. of a freshly prepared 5 per cent aqueous solution of sodium thiosulfate (filtered) and, if necessary, continue the injection up to a total of 500 cc., if possible. The solutions used in these treatments can be readily sterilized by boiling for fifteen minutes.

SODIUM NITRITE AND SODIUM THIOSULFATE TREATMENT.—Ingegno and Franco[11] treated two patients with 0.3 Gm. sodium nitrite in 10 cc. given intravenously at the rate of 2.5 to 5 cc. per minute, and followed this by 25 to 50 cc. of a 50 per cent solution of sodium thiosulfate given through the same needle and at the same rate. They warned that the sodium nitrite and thiosulfate must not be mixed before administration. If signs appeared or persisted, one-half the dose of antidotes was repeated one hour later.

From their experimental work on dogs and mice, Jandorf and Bodansky[12] stated that inhalation of amyl nitrite, administered after inhalation of low lethal doses of cyanogen chloride, was effective in saving these animals, but that this same treatment has proved ineffective in resuscitating dogs from hydrogen cyanide poisoning. The authors discuss the results of their experimentation on the basis of the probable competition between methemoglobin and tissue cytochrome oxidase for the HCN or CNCl introduced by inhalation.

CASE 1.—Three men were fumigating grapevines to kill "leafhopper," a vine parasite. The material used was calcium cyanide powder, applied by dusting machines mounted on a truck chassis. The machines throw the cyanide powder through the air to a height of about six feet and a distance of about twenty-four feet on each side of the truck, dusting two rows of vines at each trip. When calcium cyanide comes in contact with moisture, it is decomposed, giving off hydrocyanic gas, which is extremely poisonous. Dusting is usually done at night, when air conditions are quieter than in daytime. The men work in pairs and are instructed to work upwind from the section already dusted.

Details of this accident are not known, but a crew of three men was missed one night at about midnight. When found, one of the men was dead, and the other two were unconscious. First aid was given to all the men until they were removed to the hospital. The two who were still alive later developed pneumonia but recovered, with no disability.

CASE 2.—This patient was seen for the first time in September, 1945, at which time he was alleging disturbances due to an exposure to cyanide in May of 1943. He gave the following history: "I had been running a cyanide pot furnace. For some time I had noticed a cough and being unsteady on my feet. I think these were hydrocyanic fumes. One day, I got something in my mouth. I don't know what it was or if it came from the pot. I started for the drinking fountain but collapsed. I was taken to the Emergency Hospital and then to the Osteopathic Hospital at Riverside. They gave me methylene blue and some type of a hypo. At the hospital, they also gave me oxygen. I was in the hospital for three or four days and then was discharged.

"I felt weak when I came from the hospital, so I went to the beach for a while to rest. After being there about ten days, one night I got pains around my heart, had gas pains and chills and convulsions. After about three hours they managed to get a doctor, although they first got the Fire Department. I was sent to the Santa Ana Hospital where they gave me a carbogen and amyl nitrite. Then I went back to the Osteopathic Hospital at Riverside. They gave me a blood transfusion but the donor's blood was not compatible with mine. I think this experience and the beach experience are the real cause of my trouble.

"After I went home again I felt nervous, my eyes bothered me—there would be alternate light and dark spells before my eyes. I would have trouble focusing my eyes—still do. I couldn't stand noise or people. Driving a car makes me nervous—I get spasms in my diaphragm and my throat. I have difficulty in sleeping. This all sounds funny, I know. I don't want to get a phobia or become a hypochondriac. I still have trouble when I eat—my eyes don't focus, there is a tightness and constriction in my abdomen and breathing becomes fast. I used to get a buzzing in my ears, or a sensation of my ears being wet. I know that wasn't true, but it seemed so."

The patient states that he returned to work in October, 1943, at a four-hour day and has gradually increased this. He is now on a forty-hour week schedule, which is the same as the entire company.

*Past Medical History.*—(1) Incipient pulmonary tuberculosis—was on the desert for one year. (2) Contracted amebic dysentery in 1928, while in Mexico. Was troubled for one year. States that recently he has had evidence of intestinal amebae which were found by Dr. S. of Riverside, who thinks the patient has had this for several years. (3) Influenza on several occasions. Denies any serious accident or operation.

*Occupational History.*—Patient began working for this company in December, 1942. He had installed "their setup" and then worked first in the heat-treating department. The operation is that of taking low carbon steel and immersing these parts in the cyanide pot for twenty to thirty minutes, after which they are quenched. This is done in a large warehouse, approximately 250 feet by 100 feet. A middle wall divides this. He states the ventilation is good, with a bifurcated blower and suction apparatus in operation. The patient wears a gas mask when operating (loading) the pot.

*Physical Examination.*—The entire physical examination and the neurologic examination were normal.

*Laboratory Examination.*—The blood count, urine, and blood chemistry were normal; Wassermann, negative.

**Discussion.**—Whether or not this patient had actually some cyanide in the mouth at the time of the initial attack cannot be stated with any certainty, although it is doubtful. However, his subsequent bizarre complaints were certainly not due to the action of cyanide. It is believed that the patient had some innocuous substance in his mouth which he believed to be cyanide, became hysterical, and then proceeded to build up a chronic illness characterized by neurotic complaints of all types.

Considerable time was spent with this patient explaining the action of cyanide and the improbability of any aftereffects. He admitted that no one had ever explained this to him before. He was seen one month after the initial examination, at which time he was entirely free of all symptoms. It is the writer's opinion that many cases of so-called chronic illness from cyanide exposure, or the so-called sequelae, are of this type.

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# CHAPTER XII

# THE HALOGENATED HYDROCARBONS

## Methyl Bromide

Methyl bromide poisoning is not one of the industrial illnesses which is frequently encountered; yet it occurs sufficiently often to warrant closer attention than has been given to it in textbooks on toxicology. Methyl bromide has long been used as a basis for certain chemical compounds, it has been employed in the manufacture of dyes and other coloring materials and in exterminators, and, in recent years, it has been used extensively as a fumigant.

**Properties.**—At ordinary temperatures, methyl bromide ($CH_3Br$) is a colorless gas. It is noninflammable, but at elevated temperatures it decomposes, and if it is brought into contact with a flame or hot surface, it forms hydrobromic acid, a bromine.

**Tests.**—The halide lamp will, under ideal conditions, detect the presence of 50 p.p.m. of methyl bromide in the air; but at 35 p.p.m. the test is unreliable. Since the continuous exposure of workers at such a level of contamination may cause harm, this test should not be used to determine safe working conditions. Williams[1] suggests the passing of air suspected to contain methyl bromide through a quartz tube heated to 950 to 1,000° C. which will absorb the hydrogen bromide formed in a dilute solution of sodium hydroxide, in which the bromine content can then be determined by the Kolthoff-Yutzy procedure.

**Pathology.**—In those cases where absorption has been rapid and severe, the lungs are invariably involved. Pulmonary edema is the most frequent finding, although a severe bronchitis or consolidation may also be present. In cases with fatal outcome, autopsies have revealed hyperemia and edema of the lungs as well as hemorrhagic pleural effusion. Cardiac involvement is not to be expected. Congestion of the various organs has been reported, but no characteristic changes take place in methyl bromide poisoning.

Next to the lungs, the most important findings are present in the brain. The meninges and the brain are hyperemic, scattered petechial hemorrhages appear, edema is common, and authorities report ganglionic changes characterized by fatty degeneration and shrinkage. Only in rare instances is death due to these neurologic changes, but more frequently to pathologic conditions involving the lungs, while the neurologic lesions account for the signs and symptoms manifested after withdrawal from exposure.

Methyl bromide poisoning presents one peculiar phenomenon which deserves attention at this time; namely, its "delayed reaction." In 1945 the author reported[2] an episode of methyl bromide poisoning among a large group of workers and referred to recently expressed opinions regarding the cause of this delayed reaction. Since publication of that article, von Oettingen[3] collected all cases reported in the literature and included a review of the experiments which had been carried out in order to explain the reason of the delayed reaction. The following summary is taken from his excellent article:

> "It is generally accepted that the narcotic action of aliphatic narcotics such as chloroform is due to the entire molecule and is closely connected with the lipoid solubility, and that the intensity of their action is determined at least in part by their partition coefficient oil/water or their solubility coefficient in oils. That this holds true for the toxic manifestations of methyl bromide is questionable. As compared with chloroform and its higher homologue, ethyl bromide, the narcotic action of methyl bromide (that is, the depressing effect on the cerebral cortex) is comparatively slight. It is apparent that the narcotic action of methyl bromide is in accordance with its relatively low lipoid solubility, and that the toxic manifestations characteristic for methyl bromide which are not shown either by ethyl bromide or by chloroform must be due to a different mechanism. Irish, Adams, Spencer, and Rowe[4] (1941) demonstrated that in rabbits showing functional changes after repeated exposures for 8 hours to a concentration of 60 p.p.m. of methyl bromide in air the inorganic bromide level in the blood increased from a normal of 1 mg. per cent to 11 mg. per cent. However, when rabbits were fed sodium bromide in sufficient doses to produce this and even higher levels of bromide in the blood (62 per cent) they failed to show any functional response similar to that observed in animals fed methyl bromide. Similarly, rabbits exposed to methanol vapors in concentrations equivalent to or many times higher than those which would result from such concentrations of methyl bromide as used in the exposure experiments to methyl bromide failed to show a functional response similar to that produced by methyl bromide. Such negative results were also seen in rabbits exposed to 5,000 p.p.m. methanol in air and receiving at the same time daily doses of 0.1 Gm./kg. of sodium bromide, in spite of the fact that the bromide level in their blood rose as high as 90 mg. per cent. It is, therefore, evident that neither a high bromide level nor a high methanol level, or a combination of both, in the blood results in the typical picture of methyl bromide poisoning. Irish, Adams, Spencer, and Rowe[4] (1941) found that feeding rabbits 22 daily doses of 0.03 Gm./kg. of methyl bromide dissolved in olive oil produced a bromide level of 20 mg. per cent in the blood and the same type of response (paralysis) as seen after the inhalation of methyl bromide vapors, but that it required 50 daily doses of 0.30 Gm./kg. of ethyl bromide with a bromide level of 42 mg. per cent in the blood to elicit the same effect. From this they concluded that the typical response to methyl bromide is a function of the methyl bromide molecule proper. It should, however, be pointed out that according to Miller and Haggard[5] (1943) olive oil alone, given in doses up to 15 cc. daily over a period of 3 to 8 weeks, produces in rabbits loss of weight, illness, sometimes paralysis of the hind legs, and in most animals death. They also showed that rats, which tolerate large doses of olive oil better than rabbits, died within 5 to 7 hours after the oral administration of 100 mg./kg. of methyl bromide, whereas animals given as much as 1200 mg./kg. of ethyl bromide in the same way neither died nor showed any serious ill effects. It should also be pointed out that ethyl bromide has been used as a narcotic agent without causing any signs or symptoms

similar to those resulting from even less severe exposure to methyl bromide. It appears, therefore, that the toxic effects of methyl bromide are fundamentally different from those produced by ethyl bromide, and that one has to look for a different explanation for the peculiar effects of methyl bromide. Miller and Haggard (1943) refer to Harrison, Darrow, and Yannet[6] (1936), Manery and Hastings[7] (1939), and Wallace and Brodie[8] (1939) as having demonstrated that the bromide ions present in the blood stream do not penetrate the cellular membranes in appreciable quantity (except red blood cells) but that they are limited to the extracellular fluid. They pointed out that alkyl halides, such as methyl bromide, penetrate the cells and dissolve in the intracellular fluid. In case hydrolysis takes place within the cells, bromide ions are liberated within the cell where they are trapped because they pass only with difficulty through the cellular membrane and therefore their pharmacodynamic action is prolonged. They assume further that under these conditions even small quantities of bromide may produce an atypical and prolonged reaction and that for this reason the bromide level in the blood would be no indication of the extent of the bromide action. They presented experimental data to show that in rats there is a different distribution of bromide between intracellular and extracellular fluid after the oral administration of methyl bromide in oil than after the oral administration of sodium bromide in water, whereas the distribution of bromide after oral administration of ethyl bromide in oil corresponds closely to that found after sodium bromide, indicating that more bromide is retained within the cells after administration of methyl bromide than after administration of ethyl bromide or sodium bromide. As pointed out by Miller and Haggard[5] (1943) there is at present no evidence that the intracellular bromide has certain pharmacological peculiarities not yet demonstrated or that within the cell it may cause direct organic injury to the cell. They also pointed out that in case the intracellular bromide is formed from intracellular hydrolysis of methyl bromide, the other product of hydrolysis should be methanol, that for each mg. of sodium bromide 0.311 mg. of methanol should be formed, and that with a retention of a total average amount of 109.8 mg. per kg. of sodium bromide at death the corresponding quantity of methanol would be 33.1 mg. per kg., an amount which would cause no symptoms of poisoning. It appears, however, that with some justification one can also assume that the same quantity of methanol formed within the cells may have more marked effects than when distributed throughout the organism. As pointed out, the similarity between the toxic action of methyl bromide and methyl iodide on the one hand and the difference in the toxicological picture between methyl bromide and ethyl bromide on the other, point to the importance of the methyl group for the characteristic action of methyl bromide. Although the possibility of intracellular hydrolysis with the formation of bromide and methanol and its oxidation products formaldehyde and formic acid may offer one explanation of the specific toxicity of methyl bromide, it may also be possible that methyl bromide as well as methyl iodide forms a quaternary ammonium compound with certain nitrogen groups of enzymes and other biologically important amines in the organism.

"It has been shown that methyl bromide differs from other aliphatic chlorinated hydrocarbons in that it has a very weak narcotic action, but that after a latent period it produces functional and perhaps also pathological changes in the lower sections of the brain, the basal ganglia, the cerebellum, and the pyramidal tract, as indicated by the changes of the reflex excitability and visual and speech disturbances. In this action it resembles manganese and carbon monoxide, both of which may affect the oxygen metabolism in certain sections of the brain."

Dr. James H. Sterner, Director of the Laboratory of Industrial Medicine of the Eastman Kodak Company, writes in a personal communication: "On intoxications in general I have become quite convinced that the over-simplification in attributing the toxic effects to the breakdown products of a substance has been very much overdone and in most instances is probably incorrect. In many instances the intact molecule is the offending agent, and the small amounts of degradation products are relatively inconsequential. Or the mechanism of action is due to the dynamic aspects of the degradation with the interference of enzyme systems, an effect of the energy degradation and not due to the end products."

**Signs and Symptoms.**—In acutely fatal or near-fatal cases the signs and symptoms are predominantly due to pulmonary involvement. Some patients, suffering from methyl bromide poisoning, may first note headache, vertigo, nausea, or vomiting but soon thereafter pulmonary edema, cough, frothing at the mouth, rapid shallow respirations, elevation of the temperature, coma, and finally death will ensue. Convulsive seizures may detract attention from the pulmonary pathology.

In patients who have been exposed to small but nevertheless harmful amounts over a period of time, the clinical picture is bizarre and almost unbelievable. In 1944 this author examined thirty-four date packers at Indio, Calif., who had been exposed to methyl bromide for a period of several weeks. The incidence of the various signs and symptoms is to be found in Table V. It will be noted that each patient complained of a visual disturbance which frequently constituted the first sign of trouble. Mental confusion and speech disturbances likewise occupied a prominent place in the incidence of signs and symptoms following methyl bromide poisoning. All these patients recovered without suffering permanent pathologic disability. However, two developed neuroses which, at the time of the initial report, were believed to be due, in part, to the prolonged and unwarranted delay in the payment of compensation. It was later learned that one of these two claimants recovered completely, while the other, an elderly lady, persisted in alleging unfounded symptoms.

In addition to the signs and symptoms mentioned, it should not be overlooked that exposure to high concentrations of methyl bromide vapor can produce chemical burns of the skin, characterized by erythema, vesicles, or even large blisters.

**Disability.**—In the fatal cases, death usually occurs within two to twenty-four hours. From an analysis of cases of severe but not fatal poisoning reported in the literature, it can be stated that, if death does not occur within two days, the paient can be expected to recover. It must be regarded as highly questionable whether any pathologic change resulting from intoxication will be of permanent character.

TABLE V. INCIDENCE OF VARIOUS SIGNS AND SYMPTOMS OF METHYL BROMIDE POISONING
(Johnstone: Indust. Med., 1945.)

| SYMPTOMS | TOTAL | INTENSITY | | | DURATION | | | |
|---|---|---|---|---|---|---|---|---|
| | | SEVERE | MODERATE | MILD | FEW DAYS | FEW WEEKS | 2 TO 5 MONTHS | POSSIBLY PERMANENT |
| Visual disturbances | 34 | 2 | 7 | 25 | 27 | 5 | 2 | 0 |
| Speech disturbances | 23 | 5 | 8 | 10 | 23 | 0 | 0 | 0 |
| Mild mental confusion | 15 | . | . . | 15 | 15 | 0 | 0 | 0 |
| Hallucinations | 7 | 7 | . . | . | 7 | 0 | 0 | 0 |
| Melancholia | 2 | . | . . | 2 | . . | . . | 2 | 0 |
| Coma | 4 | 4 | . . | . . | 4 | 0 | 0 | 0 |
| Convulsions | 1 | 1 | . . | . . | 1 | 0 | 0 | 0 |
| Apathy | 1 | 1 | . . | . . | . . | . . | . . | 1 |
| Tremor | 4 | . . | . . | 4 | . . | 3 | 1 | . . |
| Neurosis | 2 | . . | 2 | . . | . | | 1 | 1 |
| Fainting attacks | 3 | . . | . . | 3 | . | 2 | 1 | . |
| Numbness of extremities | 18 | . . | . . | 18 | 4 | 11 | 3 | . |

**Prevention.**—Accidental methyl bromide poisoning is a decidedly unnecessary misfortune, since the preventive methods are simple. Workers should be informed of the potential danger, and adherence to protective measures should be enforced. Many plant managers are still oblivious of the fact that there is no safety in secrecy. The United States Public Health Service offers the following preventive measures:

1. Avoid breathing air containing methyl bromide.
2. On completion of fumigation, provide thorough ventilation for cars, rooms, or buildings before entering.
3. When necessary to enter space containing methyl bromide, use a gas mask provided with a canister giving protection against organic vapors, or a positive pressure hose mask (masks and canisters to be approved under the U. S. Bureau of Mines Schedules 14D or 19A. Canister black, type B).
4. Avoid spilling of methyl bromide. Get to fresh air immediately in case of spillage. Remove any clothing in contact with the skin which has become impregnated with the liquid.
5. Post warning signs, notifying that methyl bromide is being used and that the gas is toxic.
6. Containers of methyl bromide should be stored in a cool, well-ventilated place, outside inhabited buildings. Avoid leakage by seeing that valves of cylinders are tightly closed.

**Treatment.**—Victims of severe, acute intoxication should be removed from their exposure and taken into the open air, all clothing removed, and oxygen immediately administered. This oxygen therapy should be continued for at least twenty-four hours and renewed at the slightest sign of pulmonary pathology or cardiac embarrassment. Venesection has been suggested for relief of pulmonary edema. For severe convulsions, inhalations of chloroform are advisable. For the restless, the barbiturates have proved useful. In subacute cases or in those individuals suffering from a chronic exposure, the mere removal from such exposure together with general supportive measures and assurance of eventual recovery is all that is necessary.

**Case Histories.**—

CASE 1.—F. K., aged 34 years, a widow, first complained of feeling run down and tired for a period of two weeks and then reported to her physician because of double vision. She developed a paretic type of speech, could not enunciate, and then could not choose the correct words to convey her meaning. A maniacal state followed a period of hallucinations. She screamed and raved constantly that someone had killed her child and was attempting to kill her. Mental condition cleared after ten days. Three months after the acute illness this patient complains of occipital headaches, fainting attacks, pain and tension in the cervical area, and numbness and tingling of the extremities. Physical examination results in normal findings and there is no evidence of optic atrophy.

CASE 2.—H. E., a woman, aged 56 years, noticed that she was tired. Her first intimation that anything was seriously wrong came when she signed her day slip and noticed that she saw two rows of figures instead of one. Two days later her family noted that she made wild remarks and her speech became thick. She then developed hallucinations, believing there were lugs of dates stacked in her room and under her bed; that men were in her room painting these lugs. During this period she was exceedingly loquacious, although her family could not understand her, since her speech was "thick" and disconnected. Her visual disturbance persisted for two months. Four months after onset she claimed to be nervous and weak and to have frequent headaches and numbness of the lower third of each leg. Physical examination on May 16 produced nothing noteworthy.

CASE 3.—R. O. D., a man, aged 30 years, operator of a fumigating room, first noted blurred vision, then "purple" vision, and soon became nearly blind. He developed hallucinations, slurred speech, and hesitancy in the choice of his words; he was disoriented, and about five days after onset of symptoms he became semicomatose. A checkup examination three months later revealed the eye muscles weak to the right; 20/50 vision of the right eye, 20/40 of the left; color vision was inaccurate and the patient complained of eye strain—"that his eyes got tired after reading a few minutes." Otherwise he had no complaints.

CASE 4.—A. V., a Mexican, aged 38 years, first noticed blurred vision, then an unsteady gait. His wife accused him of being drunk on his return home from work. One odd complaint was that "when I want to step forward, I step backward." He eventually showed evidence of hallucinations, numbness of the legs, and weakness. These symptoms all cleared up after a period of three weeks.

CASE 5.—K. P., a woman, 55 years of age. For a period of one week her fellow workers noticed that she would make strange remarks and then, while at work, she became irrational; she picked an imaginary object from her clothes, made passes at imaginary objects in the air, claimed lights were flashing into her bedroom window, spoke German (her native tongue) for the first time in twenty years, and would pray for hours. Her gross mental abnormality cleared after three weeks, but four months after onset she still remained apathetic and listless. She complained of buzzing in the ears, a choking sensation, pains in the chest, and insomnia. Aside from a mild hypertension and a hesitant gait, physical examination carried out just prior to the present report produced nothing unusual.

In the literature, which reports cases as early as in 1899, there appear twenty-eight deaths from methyl bromide poisoning. In 1945, Clarke, Roworth, and Holling[9] described four cases of methyl bromide poisoning, two of which ended fatally. These cases resulted from a leak in the fire-fighting apparatus on a ship of the British navy. Another British investigator, Wyers,[10] reported nine cases of intoxication, among which there was one death.

### Methyl Chloride ($CH_3Cl$)

Methyl chloride finds its chief industrial use as a refrigerant, although air-conditioning workers, color and dye makers, perfume makers, and pharmaceutic workers may also occasionally be exposed to it. Barach, Yaglou, and McCord[11] have pointed out that in refrigeration, during the past few years, methyl chloride has been rather extensively substituted for Freon, due to the demand for Freon for military requirements.

**Properties.**—Methyl chloride is a colorless gas which may be easily compressed to a colorless liquid with an ethereal odor and a sweet taste. It boils at 24° C. It has a lower limit of inflammability of 8.2° and an upper limit of 18.7° C.

**Pathology.**—From the very few autopsied cases it is evident that generalized petechial hemorrhages occur especially in the pleura, the epicardium, and endocardium. Marked congestion of the lungs, the kidneys, and the liver has been noted.

**Signs and Symptoms.**—Acute poisoning does not take place frequently and is usually the result of escaping gas and a breakdown in home refrigeration systems. A mild intoxication will produce headache, vertigo, slight mental confusion, nausea, and vomiting; moderately severe poisoning will, in addition, cause more pronounced toxic manifestations of the nervous system, such as convulsions, visual disturbances, and coma.

Chronic intoxication is occasionally seen in refrigeration plant workers or repair men. In such cases, restlessness, insomnia, staggering gait, visual disturbance, tremors, and the like are noted. Anuria has been reported as well as general indications of nephritis. Anemia is not an uncommon finding.

**Tests.**—There are no specific tests. The finding of formates and acetone in the urine and also the sweet odor of the breath or an "acetone" breath are confirmatory aids.

**Medicolegal Aspects.**—

Temporary Disability.—Patients suffering minor exposures recover within a few hours to a day or two. Severe exposures may require several weeks to a month. These patients usually complain of residual headache and nervousness. Neurosis or malingering must be considered beyond that time.

Permanent Disability.—If death does not occur in the severely exposed, eventual return to normal may be expected.

**Treatment.**—Treatment is the same as for methyl bromide.

**Illustrative Cases.**—

Case 1.—While repairing a refrigerator located in a small, confined passageway of an apartment, a 32-year-old repair man noted dizziness and headache. He stepped out on the balcony for fresh air and a rest. After ten minutes he returned and continued working for nearly two hours. The owner found him dazed and stuporous; when aroused, he answered questions incoherently. She thought that the man was drunk and telephoned his employer. Wisely sensing the possibility of exposure to a gas, the employer rushed him to a hospital. Upon arrival he responded intelligently to questioning. Vomitus was observed on his clothing, and he vomited several times following arrival. The odor of his breath was like that of chloroform. The temperature was normal; heart rate, 102; blood pressure, 124/80. The general physical examination was negative, and no abnormal neurologic findings were present. The urine and blood count upon admission were normal. The following morning the urine was mildly positive for acetone and formic acid. The patient was discharged the third day after admission, at which time the red blood count was 3,400,000; hemoglobin, 60 per cent; white blood count, 12,350; polymorphonuclear neutrophiles, 68 per cent; lymphocytes, 23 per cent; large mononuclears, 5 per cent; eosinophiles, 4 per cent; the urine was normal. Checkup examinations during the next three weeks were negative. On the twenty-first day following exposure, the blood count was: red blood count, 4,700,000; hemoglobin, 80 per cent; white blood count, 8200. The patient returned to work sixteen days after exposure.

The following case is one reported by McNally.[12]

Case 2.—"A. T., found in a coma in her apartment, was removed to the Cook County Hospital by the police on Dec. 26, 1942. Upon admission to the hospital the temperature was 100° F.; pulse rate, 88; and respirations, 28. The blood pressure was 110/70. The patient's condition was critical.

"Physical examination revealed that the patient's right pupil was slightly dilated, but that the left pupil was not dilated. There was a definite marked rigidity of the neck. A slight enlargement of the heart was present, and there were râles at the bases of both lungs. A sweetish odor on the breath was noticed. The following day the patient regained consciousness, but the rigidity of the neck still remained. There was weakness of the right arm but no flaccidity. The patient was fully conscious four days after admission to the hospital. The author saw the patient on Jan. 8, 1943, and made a diagnosis of methyl chloride poisoning.

"*Laboratory Studies.*—Examination of the urine showed that it contained a trace of acetone; there was no albumin and no sugar. The blood count showed a hemoglobin of 80 per cent, 4,330,000 red blood cells, and 18,400 white blood cells with 92 per cent neutrophiles and 8 per cent lymphocytes. A blood chemistry test showed the following: nonprotein nitrogen, 60; creatinine, 1.6; and chlorides, 490. A spinal puncture revealed there was no increased pressure, but the spinal fluid contained blood."

### Dichlorodifluoromethane ($CCl_2F_2$)

This chlorine-fluorine compound is probably the safest of all refrigerants and is known commercially as Freon or F-12. It is noncombustible and nonexplosive. Experimental work indicates that ill effects from exposure to this gas are remote.

### Dichloroethane ($CH_2Cl\text{-}CH_2Cl$)

More commonly known as ethylene dichloride, dichloroethane is used as a solvent for the extraction of edible oils and fats, as a general solvent for rubber in the manufacturing of dipped goods and for some resins, mastic, wax, etc. It has an odor not unlike chloroform.

Acute exposure causes nausea, headache, dizziness, restlessness, and weakness. Severe intoxication will produce narcosis and hemorrhages into the intestines, lungs, and pleural cavity. Necrosis of the liver may result.

Symptoms resulting from chronic exposure to ethylene dichloride are not reported in the literature, although McNally[13] cites the case of two men working in the cholesterol division of a packing plant, who became ill from inhalation of this substance. Wirtschafter and Schwartz[14] studied three cases of acute poisoning in which, in addition to the symptoms mentioned, there appeared evidence of liver damage and severe irritation to the skin. It is interesting to note that because of epigastric cramps, calcium gluconate was administered. Heppel and associates[15] carried out a series of experimental studies on the influence of dietary factors in the toxic action of ethylene dichloride on rats. They found that weanling rats maintained on low choline diets were more susceptible to this substance than were control animals. When the choline diets were supplemented with methionine and choline, the mortality after exposure was greatly reduced. Weanling rats maintained on diets deficient in protein resisted the effects of repeated inhalation exposures as well as for the control animals, when adequate provision was made for methionine and choline.

### Ethylene Chlorohydrin ($CH_2Cl\text{-}CH_2\text{-}OH$)

Chemically this substance belongs in the glycol group. It is a clear, glycerine-like fluid with an odor similar to that of alcohol. It is used as a solvent for cellulose acetate, resin, wax, and lacquer and also as an addition to plasticizers for paints and varnishes.

From the literature it appears that the symptoms of acute nonfatal cases are headache, vertigo, nausea, vomiting, weakness, and irritation to the eyes. Koelsch[16] reported two fatal cases; in one of these the onset of symptoms was delayed. Post-mortem examination revealed edema of the lungs and brain, acute gastrointestinal catarrh, degeneration of the kidneys, and lesions in the heart. In Los Angeles in 1944 a fatality occurred in a local plant, and the death was investigated by Dierker and Brown.[17] In this instance, a petroleum hydrocarbon of the kerosene range had been used in a certain manufacturing process, but when the supply ran out the workman substituted ethylene chlorohydrin. After an exposure of two hours he developed headache, vertigo, and nausea. He was helped to the dispensary where he remained until the late afternoon, at which time cyanosis, labored breathing, and a rapid, irregular pulse had become evident. Despite oxygen therapy and stimulants he died about eight hours after removal from exposure. It was estimated that the working atmosphere contained 305 p.p.m. of ethylene chlorohydrin.

At autopsy the chemical analysis of the liver, lungs, and blood proved negative for volatile chlorinated compounds. The gross findings were as follows:

The heart, including the coronaries, was essentially normal as was the aorta. The lungs were edematous, with a tendency to emphysema. The liver was deeply congested, though normal in shape and structure. The pancreas and spleen were normal, as were the stomach and brain. The kidneys were deeply congested.

The microscopic findings were:

Liver: The liver showed edema and swelling of the cells and many were vacuolated. There were areas of necrotic liver cells and engorgement of the blood vessels with interstitial hemorrhages.

Kidneys: The tubules showed cloudy swelling, intense engorgement of the vessels, and some broken-down parenchymal cells in the collecting tubules.

In 1944 Goldblatt and Chiesman[18] reported both clinical and experimental observations on the toxic effects of ethylene chlorohydrin. Of the eleven clinical cases, two were fatal, one being acute while the other was what the authors termed subacute. The cause of death was not clear, but it appeared evident that ethylene chlorohydrin acted as a "violent cerebral and vascular poison." The pathologic changes in the lungs, kidneys, and liver were marked.

These two investigators concluded that every possible precaution must be taken to control the concentration of the vapors of ethylene chlorohydrin in the atmosphere "since no concentration of the compound is considered safe if exposure is a daily occurence." In respect to the preventive measures, Smyth and Carpenter[19] warn that absorption of this substance through the skin may prove fatal; that rubber gloves and other material offer little protection and that therefore industrial processes and operations must be designed to avoid any human contact with ethylene chlorohydrin or its solutions.

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# CHAPTER XIII

# THE CHLORINATED HYDROCARBONS

The solvents to be discussed in this section might also have been included in the previous chapter devoted to the halogenated hydrocarbons, since they are halogen compounds of the aliphatic series. However, because these "chloro" compounds are so widely used in industry and because a fairly frequent incidence of illness resulting from undue exposure to these solvents is being reported, they have been singled out for special consideration. Before discussing each hydrocarbon of this group individually, it is necessary to call attention to the physiologic action of the chloro compounds. In cases of medicolegal controversy and in occasional case reports, unwarranted assertions have been made regarding the effect of these substances upon the body, especially with respect to carbon tetrachloride and trichlorethylene.

Carbon tetrachloride and trichlorethylene belong to the anesthetic group of gases. They act primarily upon the central nervous system. A secondary effect of carbon tetrachloride is upon the liver and kidneys, while only occasionally will trichlorethylene involve these organs. This disturbance is practically the limit of their effect upon the organs of the body in nonfatal cases of acute poisoning. Yet the established and recognized entities of these two diseases are constantly being distorted by those who "write in" pathologic lesions without proper foundation. Within the past several years the author has noted claims of gastric ulcer, coronary thrombosis, bronchial asthma, and, of all things, acute peritonitis alleged to have been caused by exposure to either of these two solvents.

The action of the anesthetic gases upon the heart requires clarification. Ever since Levy[1] in 1911 observed the cause of death resulting from chloroform anesthesia, it has been known that the anesthetics can cause cardiac dysrhythmia. Geiger[2] carried out a clinical study and analysis of the electrocardiogram of a patient to whom carbon tetrachloride had been administered by inhalation. This patient had evidenced extreme susceptibility to both trichlorethylene and carbon tetrachloride. The tracing revealed the prompt appearance of ventricular ectopic beats, which rapidly increased to multifocal beats of such profusion that they completely dominated the cardiac rhythm, until one minute after cessation of the inhalation. Chenoweth[3] also induced ventricular fibrillation in animal experiments with certain hydrocarbons.

It is essential that this specific action of carbon tetrachloride, trichlorethylene, and some of the other hydrocarbons be thoroughly appreciated. In disturbing the rhythm of the heart, the action is typical of the anesthetic gases in which ventricular fibrillation or even cardiac arrest may occur. But this particular cardiac involvement should be distinguished from coronary heart disease. It is believed that reference in the literature to the action of the hydrocarbons upon the pacemaker of the heart has led to the erroneous assumption that any or all types of cardiac pathology can be caused by intoxication from these solvents. But because this supposition is inaccurate, it should be eradicated and corrected.

### Carbon Tetrachloride ($CCl_4$) (Tetrachloromethane)

Carbon tetrachloride is a colorless liquid with an odor similar to chloroform. Because it is inexpensive, noninflammable, and nonexplosive it is the most commonly used solvent in industry. However, although noninflammable at ordinary temperatures, it will decompose at elevated temperatures into small amounts of hydrochloric acid and phosgene. It boils at 76.7° C.

**Occupational Uses.**—The most common industrial use of carbon tetrachloride is as a degreasing agent for metal parts. It is also used extensively in the dry cleaning industry, in the extraction of fats, as a solvent for resins and gums, in fire extinguishers, and in insecticide sprays. Carbon tetrachloride is either the chief ingredient or a component of many trade name products such as Carbona, Tetra, Tetracol, Tetraform, Asordin, Spectral, and Benzinoform and of the fire extinguisher, Pyrene.

**Pathology.**—The initial acute intoxication of carbon tetrachloride is centered upon the nervous system where it exercises a narcotic action. Its secondary action is toxic and involves the kidneys and the liver. While the narcotic effect of the anesthetic solvents causes temporary derangement of various bodily functions, pathologic changes of the central nervous system are rare. If a lesion occurs in the central nervous system, the resulting impairment is usually one of the optic nerve, as evidenced by dimness of vision and curtailment of the visual field. In the nonfatal cases it has been the author's experience that other lesions of the nervous system are not encountered. Animal experimentation has revealed cerebral cortex degeneration, chromolysis, and edema of the neuroglia. But comparable findings were not present in human autopsies.

A hepata-renal syndrome results from the toxic action of carbon tetrachloride, whereby the kidneys are affected far more frequently than the liver. The renal injury is essentially a destructive process of the epithelium, causing the stroma to escape. Corcoran and associates[4] describe the nephrosis due to carbon tetrachloride as follows:

> "The renal injury which may follow exposure to toxic substances is of particular interest. Here, in contrast to acute Bright's disease, the injury is

primarily epithelial and not vascular. Clinically, however, the syndrome differs only quantitatively from glomerulonephritis in its relationships of proteinuria, pyuria, cylindruria, oliguria, hypertension, and edema. Since the nephron is the structural unit of renal function and damage within it, whether epithelial or vascular, extends to the whole nephron, the gross clinical and functional patterns of acute toxic nephrosis and acute glomerulonephritis are very similar.

"The injury probably begins with contact of the toxin and epithelial renal cell at the cell's tubular surface, where the poison is apt to be more concentrated than it is in blood or interstitial fluid. When the injury is largely to the cells of the proximal tubule, as it is with mercury or tartate, it may be that the toxins have been concentrated within the cell by active reabsorption much as is dextrose in this area. At least tubular fluid is still dilute at this point and the concentration of the toxin is probably not at a maximum. As would be expected, the more common site of injury is the epithelium of the distal tubule (e.g. carbon tetrachloride, ethylene glycol, dioxane, burns, traumatic anuria and transfusion hemolysis). Here the injury seems to depend simply on the concentration of the toxin in the tubule fluid as the result of abstraction of water from it in the proximal tubule and loop of Henle. The injured cells swell and desquamate. At first, as during diabetic coma, the tubular barrier may be functionally disorganized, so that its reabsorption is no longer selective. However this may be, the injured cells themselves may impede the flow of tubular fluid and the kidney swells, partly from their obstructive effect and partly from the interstitial reaction at their injured basement membranes. Interstitial pressure is increased and effective intraglomerular filtration pressure is correspondingly reduced, as is also the rate of renal blood flow. The swelling subsides as recovery begins; renal blood flow and filtration rate are increased. The normal basic architecture and particularly the stroma of the kidney are well preserved. Restoration to integrity is therefore possible if the initial injury is not too severe."

The liver is only infrequently affected by carbon tetrachloride, and when this involvement takes place the injury is usually of a mild nature. Acute yellow atrophy is sometimes noted. Histologically the liver may show necrosis of cells, fatty degeneration, and hemorrhage. However, despite even severe injury, the liver's ability to repair is such that the damage seldom proves to be of a permanent or even prolonged character.

The lungs have been reported as being involved by carbon tetrachloride intoxication, but this is only the case in severe or fatal cases. Thompson[5] describes consolidation of the lungs in patients overcome by carbon tetrachloride on a submarine. If, however, carbon tetrachloride comes in contact with the naked flame or if it is subjected to high temperatures, its decomposition will liberate phosgene (carbon oxychloride, $COCl_2$) which is extremely irritating to the pulmonary tissue. It is suspected that most cases of pulmonary inflammation result from the action of decomposition products of this solvent.

**Signs and Symptoms.**—Acute intoxication, if mild, is characterized by headache, dizziness, drowsiness, and lassitude. In severe poisoning there is, in addition, nausea, vomiting, abdominal pain, and diarrhea. Frequently the patient only becomes aware of an existing illness a few hours after cessation of exposure. At that time, in addition to the aforementioned signs and symptoms, an involvement of the kidneys will be evident. The urine becomes scanty and dark colored, and anuria may set in. The eyes are puffy, the ankles edematous, and the patient may become comatose. At this point, examination of the urine will show albumin, red blood cells, and casts. Blood chemistry reveals nitrogen retention; the blood pressure is elevated. If the liver is involved, signs of jaundice will often be apparent. From then on the course is typical of that of a toxic nephrosis, invariably followed by recovery. If the optic nerve has been involved, limitation of the visual fields may persist longer than other signs.

Chronic exposure may lead to headache, vertigo, loss of appetite, loss of strength, and a progressively acute sense of fatigue. In this writer's personal experience there has been no evidence of anemia. Some investigators have noted a relatively pronounced lymphocytosis. Daily contact with carbon tetrachloride may cause a scaling, fissurelike dermatitis. Farrell and Senseman[6] reported an instance of polyneuritis due to absorption of carbon tetrachloride through the skin, characterized by weakness, numbness, pain in the extremities, and diminution of the deep reflexes. Complete recovery occurred.

**Laboratory Tests.**—Fujiwara[7] discovered that when sodium hydroxide (10 per cent) and a small quantity of chloroform are heated together, a red coloration develops in the upper pyridine layer. This test is not specific for any particular one of the hydrocarbons, yet it does indicate the presence in the tissues of various hydrocarbons. In 1945 Webb, Kay, and Nichol[8] devised an improved visual test for carbon tetrachloride, based on the Fujiwara reaction, which permitted the detection of concentrations as low as 0.0000021 Gm. per milliliter when dissolved in acetone. Barrett and associates[9] evolved a visual test for the detection of carbon tetrachloride by using sodium hydrosulfide.

In 1944 Maclagan[10] described the thymol-barbitone test or, as it is sometimes called, the thymol turbidity test which he associates with liver dysfunction. This was subsequently appraised by McCord, Sterner, Kiline, and Williams,[11] who summarized their investigation as follows:

> "The thymol-barbital test (thymol turbidity test) has been applied in an experimental investigation of animals (rabbits—rats) directed to the establishment of the first demonstrable evidences of organic damage from carbon tetrachloride after its inhalation in vapor form.
>
> "At three concentration levels (40, 60 and 80 parts per million) some increases in thymol turbidity have appeared at the end of five days' exposure, with further increases at the end of ten days' exposure (twelve days' elapsed time).
>
> "Injurious action, if any, at lower concentrations than 40 parts per million has not been explored.
>
> "The increase in thymol turbidity arising in the course of exposure essentially disappears by the end of seven days after the termination of exposure.

"Evidence of injury other than thymol turbidity from carbon tetrachloride in concentrations of 40 parts per million has been secured for rats, and for both rats and rabbits from higher concentrations.

"The earliest demonstrable effects in animals induced by relatively low concentrations of carbon tetrachloride appear to be a variation in phospholipid metabolism as measured by the thymol-barbital turbidity test. The association of the continued or more severe degrees of test reaction with well defined organic injury suggests that the responses at the above levels are indicative of incipient organic damage."

In 1938 Hanger[12] proposed a simple test by which disturbances of the liver parenchyma may be detected through the capacity of the blood serum to flocculate a colloidal suspension of cephalin-cholesterol emulsion. The reactions are expressed as 1 plus, 2 plus, 3 plus, or 4 plus. Kolmer's[13] directions for the cephalin flocculation test (Hanger), which should be made on the fasting stomach, are as follows:

"1. Prepare the reagent by dehydrating finely divided sheep brain by three extractions with acetone. Dry the tissue and grind into a powder. Extract the powder three times with peroxide-free ether. Concentrate the ether extracts *in vacuo*. Add four volumes of absolute ethyl alcohol. Decant and collect the precipitate. Dissolve the latter in a minimum amount of ether. Precipitate the cerebrosides by chilling and centrifuging the ether solution. Decant the supernatant ether to which add four volumes of absolute ethyl alcohol, chill and filter. Wash the precipitate with absolute alcohol followed by acetone. Dry the precipitate (cephalin) in air and keep in a dessicator in a dark place.

"2. Prepare a stock solution by dissolving 0.1 Gm. of cephalin and 0.3 Gm. of cholesterol in 8 cc. of Squibbs anesthetic ether. Add 1 cc. slowly and with stirring to 35 cc. of freshly distilled water heated to 65 to 70° C. Raise to 100° C. and boil gently until the volume is reduced to 30 cc.; allow to cool (reagent).

"3. In a small test tube place 0.2 cc. of patient's serum (which is not over 24 hours old and has been kept in a refrigerator); add 4 cc. of 0.85 per cent sodium chloride solution in distilled water and 1 cc. of the reagent. Shake thoroughly. Keep at room temperature and read after 24 to 48 hours.

"4. Positive reactions are indicated by the formation of precipitates which may be recorded as follows: O (negative); ± (slight flocculation); + (slight flocculation); ++ (moderate flocculation); +++ (marked flocculation); ++++ (complete flocculation with clear supernatant fluid)."

Other liver function tests are too well known to necessitate their repetition here, such as the icterus index, the van den Bergh reaction, etc. Nor is it requisite to mention the routine tests to be carried out for evidence of toxic nephrosis. The presence of hemoglobin is easily detected chemically, while lesser degrees of hemolysis can be noted by staining the centrifuged urinary sediment for hemosiderin in macrophages.

**Possible Disability.**—Temporary disability from mild intoxication is of short duration, lasting a few days to a couple of weeks. Severe, nonfatal intoxication in which either the kidneys or the liver or both organs are involved will require several weeks to several months for complete recovery. No cases of permanent disability are known to this writer.

**Treatment.**—In the prevention of carbon tetrachloride poisoning, the need to change the present accepted limit of maximal allowable concentration will probably have to be considered. A number of investigators recommend that the level which is now 100 p.p.m. be changed to 50 p.p.m. Suggested protective procedures will be found in the section on industrial hygiene.

In the treatment of acute intoxication, one warning must be made: In emergency cases where cardiac collapse is suspected, it is almost a routine procedure to administer epinephrine. *This should not be done* in instances of carbon tetrachloride poisoning. It is generally recognized that if an anesthetic gas is to produce ventricular fibrillation, an elevated circulating concentration of epinephrine is required. Chenoweth[3] confirmed this fact in animal experimentation by using hydrocarbons, and arrives at the statement that the administration of epinephrine to victims of accidents who have been exposed to vapors of hydrocarbons would seem to be a particularly hazardous procedure.

No specific treatment for carbon tetrachloride poisoning has been developed. In 1944 Beattie and associates[14] reported a case of carbon tetrachloride poisoning due to ingestion and treated by methionine which they considered "to have prevented permanent liver damage." Following their report, Eddy[15] in 1945 described the use of methionine in carbon tetrachloride poisoning. In one case he administered a dosage of 2 Gm. every four hours. In another group, methionine by mouth or indwelling duodenal catheter was given in doses ranging from 6 to 12 Gm. daily.

The reports cited in the foregoing have led to the general assumption that methionine is practically specific in the prevention or repair of liver damage. Such belief, however, has not been supported by the observations reported by Higgins and his associates[16] as well as by Hobbs and Campbell.[17] In fact, Hobbs and Campbell have presented evidence to the effect that marked hepatic insufficiency may be a very serious contraindication to the parenteral administration of amino acid mixtures.

The basis upon which the methionine therapy has been developed is the belief that the depletion of body proteins induces greater susceptibility to the toxic action of certain liver injurants. A corollary belief is that the administration of protein to animals suffering from such a deficiency will reduce any injury to the liver resulting from hepatoxic agents. Miller and Whipple[18] claim that the intravenous administration of methionine, up to four hours after chloroform anesthesia, successfully protected a protein-depleted dog from fatal liver injury. It appears that the value of protein feeding resides in the sulfur-containing amino acids, methionine and cystine. In 1946 Shaffer, Carpenter, and Moses[19] conducted animal experiments to determine the value of methionine therapy and arrived at the conclusion that these animal studies failed to demonstrate any beneficial action of methionine.

The author is unable to contribute an opinion derived from his own experience, since he has never used methionine. It would seem that the consensus of opinion is to the effect that no harm comes from its use. However, in the writer's experience death does not result from hepatitis, and in the nonfatal cases no permanent liver damage is to be expected. Concerning the general treatment, the following regime is recommended.

The severe case of carbon tetrachloride poisoning will require a rigid regimen, the purpose of which is twofold: to *treat the liver and renal damage* and to *restore a normal blood chemistry.* The patient should be removed from all contact with the substance. He should be supplied with an abundance of fresh air and with inhalations of from 5 to 7 per cent carbon dioxide in oxygen, if necessary, for respiratory stimulation.

INCREASING BLOOD CALCIUM LEVEL.—These patients have a low blood calcium level. Therefore, a high calcium intake should be provided to bring their blood calcium to the high or normal level, which it has been shown experimentally prevents liver and renal damage to some degree. This can be accomplished by giving 10 cc. of a 10 per cent solution of calcium gluconate intravenously two or three times daily, with 12 Gm. calcium gluconate, or 8 Gm. of calcium lactose by mouth.

PROTEINS IN DIET.—The diet itself should contain a high proportion of carbohydrates, 200 to 250 Gm. or more per day, together with a low fat and a low *meat* protein ratio. The *meat* protein is limited because of the evidence that increased retention of guanidine prolongs and increases the symptoms of carbon tetrachloride poisoning. However, in view of the demonstrated value of protein in liver regeneration, a high level of other forms of protein should be maintained. Actually, following the early acute symptoms, a still higher protein level would probably be of value. Bread, milk, sugar, and Karo syrup are obviously of much value in the diet, to which may also be added extra vitamins.

DEXTROSE SOLUTIONS.—Intravenous administration of from 2,000 to 3,000 cc. daily of 10 per cent dextrose in physiologic saline is indicated. In *renal disease* or when *circulatory collapse* induces defective renal function, the injection of physiologic solution of sodium chloride may occasionally produce or aggravate acidosis. This effect is brought about chiefly by diluting the bicarbonate already in the body at a time when the kidneys are not excreting the excess of chloride. In some instances, then, the use of 10 per cent dextrose in distilled water, rather than in

0.9 per cent sodium chloride, may be indicated. However, in most instances the 0.9 per cent sodium chloride solution may be used since even abnormal kidneys excrete salt solution well if serum proteins are not reduced or cardiac failure is not present. Dextrose solutions, by providing food and aiding the circulation, help the kidneys to adjust extracellular volume and concentration when sufficient sodium chloride is available.

The administration of from 50 to 200 cc. of 50 per cent dextrose intravenously per day is also of value, but where *cardiac involvement* is present, not more than 50 cc. should be given at one time. It has been suggested that some of the fluids may be given per rectum, rather than intravenously; 120 cc. of 10 per cent dextrose every four hours may be used rectally.

INSULIN.—From five to ten units of insulin three times per day may aid in the oxidation of the intermediate products of protein, fat, and carbohydrate.

DIGITALIS.—This has been advised routinely for the treatment of myocardial involvement but would seem to be of questionable value in most cases. When used, regular digitalizing dosages of 1½ grains (0.1 Gm.) per 4.5 kg. of body weight should be employed. *Epinephrine should not be used.*

DIURETICS.—Diuretics, other than the mercurial ones, are indicated. In treating oliguria or anuria the use of papaverine hydrochloride or sulfate, ½ grain (0.0325 Gm.) intravenously or by mouth, is said to be of value on the basis that these drugs relax the arteriolar spasm associated with the toxic nephritis. There are claims of success for the use of hexylresorcinol for treatment of kidney irritation.

OTHER MEDICATION.—Free catharsis is of value, but oily preparations are contraindicated. If any *bronchial conditions* develop, inhalations of compound tincture of benzoin and pineneedle oil, followed by a cough syrup with codeine phosphate or sulfate added, should be used. Sedation may be necessary for the *restless* or for the excited. When the *eyes* are involved, their treatment should be consigned to the ophthalmologist.

Experimental work with a purified concentrated hog's liver extract[20] has been demonstrated and accelerated healing of liver tissue in rats after carbon tetrachloride poisoning, in addition to protecting the liver from damage. The active ingredient appeared to be sodium xanthine.

**Preface to Case Histories.**—Between 1938 and November, 1946, thirty-eight cases of carbon tetrachloride intoxication were seen by the author, along with twelve cases of alleged poisoning. In the twelve alleged cases there either existed no exposure or the exposure was so mild as not to constitute an actual poisoning. Of the thirty-eight actual cases, twenty-nine of the patients evidenced kidney damage without any liver involvement; five proved to have suffered both liver and kidney damage, while the remaining four evidenced liver impairment only. At some stage during the period of acute intoxication, eleven patients complained of disturbed vision, but these symptoms cleared without permanent sequelae.

According to the author's experience there is no doubt that, following the initial narcosis, carbon tetrachloride affects primarily the kidneys and much less frequently the liver. All other organs with exception of rare instances where phosgene is found to have affected the lungs will escape damage. It is, therefore, disconcerting to read of an occasional case of alleged carbon tetrachloride poisoning in which various organs of the body are said to have been involved. Such reported instances are open to the suspicion that either the observations are faulty or that the solvent indicated contained other ingredients besides carbon tetrachloride.

It is in this respect that the author cannot agree with certain observations found in *Toxicology and Hygiene of Industrial Solvents** by the two German writers, Lehmann and Flury. With regard to carbon tetrachloride they state: "With chronic effect there is fatty degeneration of the cardiac muscle and paralysis of the vagus nerve," but the statement is made without reference to any substantiating evidence. This startling opinion seemed to call for a careful search through the American and British literature for a possible confirmation of the alleged observation, but reference to such effects was nowhere to be found. It must, therefore, be concluded that the statement is not in keeping with the experience of other investigators.

The following illustrations are typical of the signs and symptoms of carbon tetrachloride intoxication and of the lamentable absence of even ordinary precautions in handling this solvent.

CASE 1.—In October of 1946, a small Los Angeles company contracted for the conversion of a number of Army airplanes for civilian use. A 24-year-old girl was assigned to wash down the inside of the wall in one of the planes with a sponge and a solution of "tet." No exhaust system was in operation, and she was furnished no respirator. On the first day, the girl began this work at 3 P.M. and left the plant at 5 P.M. On her way home she noticed that her head seemed "full"; she was dizzy and her mother observed that she seemed "giddy and foolish." The following morning the patient felt no ill effects and went back to her job. On this second day she worked in a small compartment in which the only ventilation was a narrow opening into the main cabin. At noon, upon emerging into the fresh air, she suddenly felt limp and began to stagger. Within half an hour she complained of severe headache. She was taken home and on arrival vomited several times. That night the headache was unbearable, the bed "kept swinging around like a merry-go-round," and vomiting continued.

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* Lehmann, K. B., and Flury, F. (editors): Toxicology and Hygiene of Industrial Solvents (translated by King, Eleanor, and Smythe, Henry, Jr.), Baltimore, 1943, Williams & Wilkins Co., p. 150.

On the morning of the fourth day her mother noticed that the patient's face was swollen, at which time a doctor was finally called. His examination revealed edema of the face and ankles, heart rate of 100, blood pressure of 160/100, and albumin and casts in the urine which was scanty and dark colored. She was hospitalized. On the sixth day after exposure, jaundice appeared for the first time. It was then noted that the liver was enlarged and mildly tender.

*Laboratory Study.*—Icteric index, 370; urinary bile, 4 plus; urinary albumin, 4 plus; N.P.N., 110; hangar cephalin flocculation, 4 plus; microscopic urine, albumin casts and red blood cells.

**Comment.—**

The patient received only supportive treatment as outlined previously in this chapter. Three weeks after the onset of intoxication she was discharged from the hospital, her complaints having completely subsided. The physical examination at time of discharge produced entirely normal findings except for a slight enlargement of the liver. The laboratory findings were within normal range with exception of the cephalin flocculation, which was 2 plus. Six weeks after onset of her illness this girl returned to work but not to the same occupation.

Case 2.—A 52-year-old workman consulted a heart specialist because of signs and symptoms typical of coronary heart disease, a diagnosis which the specialist substantiated on the basis of an electrocardiogram. The patient did not relate his occupation to his cardiac trouble, but in his occupational history he mentioned that he was engaged in a process which employed carbon tetrachloride. This started a train of thought in the doctor's mind, for he recalled that a few months previously he had read in the *Journal of the American Medical Association* an article about carbon tetrachloride and heart disease. He informed his patient that the carbon tetrachloride might have caused the heart trouble. Naturally, the workman made a claim before the Industrial Accident Commission of California, for permanent disability arising out of and in the course of employment. At the time of the hearing the claimant presented a letter from the heart specialist which Concluded "that it [coronary thrombosis] did not appear before his toxemia but only appeared after this toxemia [which] would indicate it resulted from the work entailed."

Among the several erroneous assumptions which were made in this case, two deserve special attention. First, it was revealed that the article which the doctor had recalled seeing in the medical journal was Geiger's [2] dealing with cardiac dysrhythmia. Nowhere in that article is any implication made to the effect that carbon tetrachloride causes coronary heart disease. Second, the patient never suffered any toxemia or poisoning from this solvent. The process in which he was engaged was conducted in the open yard and the carbon tetrachloride was contained in a closed receptacle. An analysis of the air taken at the edge of the cover revealed only 10 p.p.m. of carbon tetrachloride.

Case 3.—This case resembles the previous one. Most of its details are irrelevant except for the fact that the workman in question was a turret lathe operator who developed an acute coronary attack. He alleged the attack to be due to chronic exposure to carbon tetrachloride. In order to obtain a smooth, highly polished finish of the precision instrument on which he was working, the operator would squirt carbon tetrachloride onto its surface as it was nearing completion. At this level of the operation an adequate exhaust removed the vapors. Air analysis carried out at the breathing level of the workman showed 25 p.p.m. This claim was allowed by the Industrial Accident Commission upon the testimony of a physician who referred to an article in a medical periodical published by the Navy, in which several cases of carbon tetrachloride poisoning were reported.

But here again the article in question did not imply that coronary heart disease resulted from chronic exposure to carbon tetrachloride. The present case and Case 2 illustrate clearly the danger of undermining occupational disease entities by an illogical approach to the diagnosis. The correction of such a situation will only be brought about when undergraduate and post-graduate medical education includes adequate instruction in occupational diseases.

CASE 4.—P. C., a 27-year-old woman, took a job in April, 1944, as an assistant to a metallurgist. Her work required her to dip a sponge into a small open dish of carbon tetrachloride occasionally. There would be some days when this was not necessary and on other days she would use the carbon tetrachloride several times a day. Two months later, June of 1944, she noted increasing fatigue and periodic jaundice. On occasions the jaundice would be deep, while at other times it was barely visible. However, she sought no medical aid until May of 1945, one year later.

In June of 1945, an exploratory operation was performed which revealed a markedly enlarged, nodular cirrhosis of the liver. Following a short period of recuperation from the operation, the patient returned to her same employment and continued working for another four months, at which time she was forced to stop working because of extreme weakness, marked jaundice, and increasing weight. The patient died in April, 1947. The post-mortem examination revealed a cancer of the liver.

In reporting this case the writer has omitted the laboratory study which was very complete but unessential for the purpose of this report. Prior to death the patient made claim before the Industrial Accident Commission for "a carbon tetrachloride poisoning." In duplicating her exposure and estimating the possible concentration in the working atmosphere, it was the opinion of the writer that her exposure had been too minimal to produce any liver damage. Further, he has never seen a case of carbon tetrachloride intoxication where the liver became enlarged and remained so. It has been observed that in the initial stage of an acute hepatorenal involvement due to this solvent the liver would be enlarged for a very brief time. On the other hand, every case of actual liver damage due to carbon tetrachloride has resulted in a small, contracted liver—an acute yellow atrophy.

Subsequent investigation revealed that this patient had for five years prior to her employment suffered "biliary colic," pain at the angle of the right scapula, and periodic jaundice and had been studied for "gall bladder disease."

CASES 5 and 6.—Three elderly men were assigned to cleaning the grease from a storeroom and were provided rags, brushes, and open containers of carbon tetrachloride. The room was provided with no ventilation other than one door which the men kept closed because it was raining. During the first day they would seek fresh air at intervals due to the presence of dizziness and headache. The following day they worked only four hours, at which time they completed the job. All three of these men complained to their employer that they felt drunk.

During the next several days two of these men became acutely ill, but no doctor was called until the fourth day after exposure, at which time the physician found these men in a state of what he believed to be "borderline" uremia. He did not know of the exposure to carbon tetrachloride but was aware that both of these men were heavy drinkers. He also obtained the information that both of these workers, who lived together in a boarding house, had been on a three-week alcoholic spree just prior to their acute illness.

On the morning of the sixth day, A. S. died and was brought to the Los Angeles Coroner's office for autopsy. This revealed a marked nephrosis with slight evidence of liver damage. On the evening of the sixth day the other worker died and post-mortem examination revealed almost identical findings.

These two deaths were directly due to the ignorance or carelessness on the part of the employer who assigned these men to work under conditions which failed to protect them fully against the toxic properties of the solvent, carbon tetrachloride. In another instance, a nationally known department store employed a woman to "spot" clothing with carbon tetrachloride. After some weeks of doing this she reported to the writer with evidence of kidney disturbance. Inquiry showed that she was doing this work in a small cubbyhole with no ventilation.

It is cases of the type just reported that emphasize the fact that industrial hygiene is usually effectively practiced in the large, so-called dangerous industries but is ignored in the small shops or stores where dangerous practices are not suspected or not supposed to be in operation.

### Trichlorethylene ($CHClCCl_2$)

Trichlorethylene is a colorless liquid with a chloroform-like odor which boils at 88-90° C. It vaporizes easily at ordinary temperatures, is relatively stable in air, and is noninflammable. However, in contact with the naked flame it gives rise to phosgene (see discussion on carbon tetrachloride).

The industrial applications of trichlorethylene are too many to be mentioned individually, but its most common use is in degreasing, dry cleaning, and painting and enamelling, in the extraction of fats, oils, and resins, in the boot and shoe industry, in printing and photography, and as an insecticide.

**Pathology.**—The primary action of trichlorethylene is upon the central nervous system. It is not uncommon for workers exposed to excessive amounts of this substance to be rendered suddenly unconscious. Fortunately, the victims usually recover without any stigmas. Browning[21] reported on thirty-nine patients among whom unconsciousness occurred in thirty-one and semiconsciousness in six instances, while two patients were less severely affected. Three terminated fatally, but all others recovered completely. In trichlorethylene poisoning, death is due either to traumatization of the respiratory center or, more likely, to involvement of the heart, where ventricular fibrillation and cardiac arrest take place. Human autopsy material reveals insufficient evidence upon which to postulate the presence of organic injury. In cases where pulmonary irritation is present, this is believed to be due to the action of decomposition, such as hydrochloric acid or phosgene.

Trigeminal paralysis accompanying trichlorethylene poisoning is a very debatable question. The older literature claimed such an involvement, but English and American investigators deny this aftereffect to acute as well as to chronic poisoning. Optic nerve lesions, such as retrobulbar neuritis or scotomas, have been attributed to "tri" poisoning.

The kidneys or liver are not commonly injured by trichlorethylene, although this may occur as illustrated by one case from the writer's own experience. The skin is generally involved when close contact with this substance has taken place and cases have been reported where it is believed that systemic injury occurred upon absorption by the skin alone.

**Signs and Symptoms.**—Acute poisoning is characterized by headache, dizziness, giddiness, disturbed gait, and the general picture of drunkenness. The onset of these symptoms, however, may be delayed several hours. Severe exposure is able to cause unconsciousness. Following a return to consciousness the patient will complain of headache, drowsiness, apathy and various complaints consistent with any other postanesthesia state. However, such disorders are of short duration and prolonged residual disturbances are not to be expected. Whenever such complaints are alleged it should be suspected that the patient is manifesting symptoms of a neurosis. Inflammation of the skin subsides rapidly except in rare instances, as will be demonstrated in one of the author's cases. If the circumstances of exposure are such as to permit the decomposition of trichlorethylene, phosgene may produce profound pulmonary pathology (see Carbon Tetrachloride).

Because trichlorethylene does not exercise any cumulative action, it is the belief of most authorities that organic disturbances do not take place following chronic exposure, although such symptoms as general malaise, lethargy, loss of appetite, etc., may be noted. In 1931 Stuber* in Germany reported 284 cases of "tri" poisoning, and of these cases she classified 182 as chronic. But Hamilton points out correctly that these cases were "symptom cases" alleging a wide variety of complaints such as paralysis of cranial nerves, epiloid seizures, functional neuroses, bizarre psychic phenomena, anemia, gastrointestinal disorders, and many others, making the list much too all-inclusive to be accepted as reliable. Ethel Browning, in citing British experience, refutes such contentions and feels that in Stuber's cases some impurity must have been present in the substance which was called trichlorethylene.

There also seems to exist ample proof that trichlorethylene has no specific effect upon the trigeminal nerve, as was once believed, a supposition which originated in the description of such lesions by Plessner[22] in 1916. But as Hamilton and the writer[23] pointed out, subsequent investigation would deny Plessner's theory. Eichert[24] likewise contends that prolonged exposure to this substance did not injure the fifth nerve and preceding him Glaser[25] made the same denial.

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* Stuber, K.: Gesundheitsschadigungen bei der gewerblichen Verwendung des Trichlorathylens und die Moglichkeit ihrer Verhutung, Arch F. Gewerbepath. u. Gewerbehyg. 1931, II, p. 398.

**Laboratory Tests.**—Tests applicable to the chlorinated hydrocarbons in general have been cited in the discussion of carbon tetrachloride. Habgood and Powell[26] described the following test which differentiates between chloroform, carbon tetrachloride, and trichlorethylene:

"After diluting the blood sample with water, the chlorohydrocarbon is removed by steam distillation and about 40 cc. of the distillate are collected in a small measured volume of toluene. This is shaken and the toluene layer pipetted off, 1 cc. of which is added to 10 cc. of pyridine and 5 cc. of 20 per cent sodium hydroxide. After heating and cooling, the pyridine layer becomes purplish red in the presence of carbon tetrachloride or chloroform, and orange-red with trichlorethylene. The color intensity is measured, and read from a calibration curve. Quantities of these three substances varying between 2 and 0.02 mg. can be recovered from blood with an accuracy within 5 per cent."

**Possible Disability.**—Temporary disability is short, recovery occurring in most instances within a few days. Permanent disability is not to be expected.

**Treatment.**—The prevention of intoxication is relatively simple, if proper protective procedures are followed. Tanks and containers should be equipped with exhaust systems, individual protection is sometimes necessary, and the skin should also be covered. If susceptibility or addiction is suspected, workers should not be placed in contact with trichlorethylene consecutively, but rather on a rotating schedule. Care must be exercised to prevent this solvent from coming in contact with the naked flame.

In treating the acutely ill or unconscious patient it should again be emphasized that adrenalin is not to be used as a stimulant for reasons previously expressed. Otherwise, the following treatment is suggested:

RESPIRATORY STIMULANTS.—Respiratory stimulants are needed. The most valuable of all will be inhalations of a mixture of from 5 to 7 per cent carbon dioxide with oxygen. When such inhalations are not available, artificial respiration by the Schaefer method must be used, and later, if pulmonary edema is present, the administration of oxygen either by a tent or intranasal apparatus will be of value. The intravenous or intramuscular injections of coramine, 1.5 cc., or the intramuscular injection of metrazol, from 1½ to 4½ grains (0.1 to 0.3 Gm.), are of questionable value in treatment of the respiratory failure, although their use has been advised.

DIET.—Following the very acute period a soft diet and normal fluid intake may be resumed, but if symptoms of nausea and vomiting persist, the use of from 2,000 to 3,000 cc. daily of 10 per cent dextrose in physiologic saline is advisable. The administration of thiamine chloride (Vitamin $B_1$) in dosages from 30 to 60 mg. daily, at first parenterally, is suggested in treatment of the neurologic manifestations. During the convalescence a high caloric diet with vitamin adjuncts should be given.

EXPOSURE TO PHOSGENE.—If the acutely ill patient has been exposed to phosgene resulting from the decomposition of trichlorethylene by gas flames, he may present a somewhat different picture, with pulmonary edema, dyspnea, and cyanosis playing prominent parts in the picture. Here, again, respiratory and circulatory stimulants are indicated. In addition, venesection of 500 cc. may be of value in treating the pulmonary edema. During World War I, intratracheal medication of from 1 to 3 drachms of the following mixture two or three times per day was found to be beneficial after the very acute stages had passed: 5 per cent each of guaiacol, camphor, and menthol in liquid petrolatum or olive oil. No instances of lipoid pneumonia from the use of this preparation are mentioned.

THE GRAY STAGE.—During the acute phase a syndrome may occur, referred to in the findings of the chemical warfare service as the "gray stage," in which pronounced pulmonary edema, almost utter inability to breathe, and shock are seen. In such a stage the temperature falls, and there is a marked increase in blood concentration necessitating oral and intravenous administration of physiologic saline solution. A fine degree of medical judgment is needed to balance the necessity of venesection on the one hand and for decrease in excessive blood concentration by administration of fluids on the other.

CASE 1.—Using a substance known as Perm A Chor (which is trichlorethylene) in a degreasing tank into which were dipped small statues, athletic prize cups, and the like, a 50-year-old man became acutely ill. He had been engaged in this work for four weeks prior to the onset of the illness. An investigation revealed that for the last three days of employment the vaporizing coils in the degreasing tank had not been functioning, thereby permitting the escape of the vapors to the breathing zone of the patient.

The patient first noticed a loss of appetite, dizziness, and a rash over the body. The following day he vomited and noted that the face was swollen. At noon of the last day of work he felt so "drunk" that he had to go home. He did not report to a doctor until four days after he had been forced to cease working. At that time he was admitted to the Los Angeles County Hospital. He complained of dizziness, headache, and skin eruptions. No abdominal distress was present, as well as no complaints referable to the kidneys, eyes, or the peripheral nervous system.

The past medical history was entirely negative, except for the one pertinent fact that while in India with a circus he had suffered an attack of malaria accompanied by severe jaundice.

*Physical Examination.*—The negative findings will not be mentioned except to point out that the eyes, fundi, and visual fields were normal. Likewise the neurologic examination produced only negative data for evidence of motor or sensory disturbance to the peripheral nerves. The skin of the face, neck, chest, back, abdomen, arms, and legs was covered with fine vesicular erythematous eruptions which subsequently subsided into a dry, scaling dermatitis. In the initial stages the patient complained of intense burning and itching of the skin.

Although the laboratory findings indicated a toxic disturbance of the liver, an enlargement of that organ could not be palpated, nor was any liver tenderness present. The kidneys, spleen, and abdomen were negative to palpation. On admission the temperature was 100°; blood pressure, 125/80; pulse, 100.

TABLE VI. LABORATORY STUDIES

| DATE | ICTERIC INDEX | A/G RATIO | PROTHROMBIN (%) | N.P.N. | CEPHALIN FLOCCULATION | | THYMOL FLOCCULATION | FUJIWARA TEST |
|---|---|---|---|---|---|---|---|---|
| | | | | | 24 HR. | 48 HR. | | |
| 6/5/46 | 13 | 4/2.4 | – | – | – | – | – | – |
| 6/11/46 | – | – | – | – | – | – | – | ++++ |
| 6/13/46 | 40 | 3.3/2.5 | 28 | 27 | – | – | – | – |
| 6/17/46 | 27 | 3.8/4.1 | 100 | – | +++ | ++++ | 9 | +++ |
| 6/21/46 | – | – | – | – | – | – | – | ++ |
| 7/5/46 | 6 | 3.9/3.9 | – | – | – | – | – | – |
| 7/9/46 | – | – | – | – | ++ | – | 4 | – |
| 7/18/46 | 4 | 4.6/3.1 | 100 | 28 | ++ | +++ | 2 | – |
| 7/20/46 | Discharged from hospital with no symptoms; skin rash present but subsiding | | | | | | | |

CASE 2.—Two Negroes were assigned the task of cleaning machinery parts in a large tank containing trichlorethylene. One of these was but slightly exposed since he was required to carry and stack the parts after they had been cleaned. The other man worked over a tank of this solvent in the far corner of the plant where ventilation was poor. This was the first day this work was undertaken, and after about two and one-half hours, the Negro engaged in the actual cleaning fell over onto the floor unconscious. Rushed to the hospital, he arrived in a semicomatose state. He could be aroused but could not answer questions. There was a two-inch gash over the right temporal area. The patient's partner described the work these two had been doing and volunteered the information that the patient complained of being dizzy and "acted drunklike." Further examination disclosed profuse perspiration; deep, slow respirations, which were increasing in frequency as the examination proceeded; heart rate of 86; blood pressure, 102/70. While the examination was being conducted, the plant foreman was reached by phone and after slight delay gave the information that the solvent was trichlorethylene. Oxygen was then administered. The patient became mentally clear within one-half hour, only to vomit frequently for the next several hours. During this time he complained of severe headache and epigastric pain. Subsequent laboratory investigation produced no significant facts. He was discharged from the hospital in three days and returned to work at the end of ten days, free from symptoms.

## Tetrachlorethane ($CHCl_2CHCl_2$)

This solvent is noninflammable and has an odor similar to chloroform or carbon tetrachloride. In the presence of moisture it liberates hydrochloric acid. Its specific gravity is 1.6.

Tetrachlorethane is the most toxic of the chlorinated hydrocarbons and there exists no necessity for its industrial use. It was, however, widely used in World War I. It possesses all the properties of a solvent agent which have been mentioned in discussing the other solvents. Because of its toxic potentialities it is not used any longer in industry to any significant extent.

It appears that the substance is absorbed slowly and that its action is prolonged. The actual acute illness is generally preceded by prodromal, vague distress extending over a period of time and followed by the sudden onset of severe toxic symptoms. After a period in which headache, loss of appetite, fatigue, and gastrointestinal disturbances are experienced, jaundice occurs at first without signs of toxemia. But this stage is followed by another one in which toxemia accompanies the jaundice. According to Minot and Smith,[27] and as later confirmed by Parmenter,[28] the jaundice is hepatogenous and not hemolytic, and for that reason no appreciable anemia or change in the blood picture occurs.

In fatal cases the post-mortem examinations show generalized hemorrhage into most of the organs of the body, together with edema of the brain. Acute cirrhosis and atrophy of the liver is commonly encountered.

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# CHAPTER XIV

# CHLORINATED NAPHTHALENES AND DIPHENYLS

The chlorinated naphthalenes are compounds in which one or more of the hydrogen atoms of naphthalene have been replaced by chlorine, forming a series from monochloronaphthalene to octochloronaphthalene. A common mixture is trichloronaphthalene which is derived from tri and tetra and contains approximately 50 per cent chlorine. Another common mixture consists of tetra-, penta-, and hexachloronaphthalene in combination with chlorinated diphenyls. The chemical origin of these mixtures is naphthalene, a colorless, crystaline solid which melts at 80° C. and boils at 217° C. It has an empiric formula of $C_{10}H_8$.

In the manufacture of chlorinated diphenyls, $C_6H_6$ is converted into $C_{12}H_{10}$, which in turn is chlorinated into $C_{12}H_{10}$. The higher the chlorination, the higher the toxicity. Also the higher the chlorination, the higher the melting point of the artificial wax and therefore the more waterproof and heat resistant the substance. The chlorinated naphthalenes are too often referred to as Halowax, a trade name of the Halowax Corporation. This indiscriminate use of a trade name is regrettable since it does not disclose the chemical nature of the substance.

**Occupational Exposure.**—The most widespread application of these substances is in the electrical industry, where they are used to impregnate cable coverings, as well as wires and radio condensers. To a much lesser extent they serve as plasticizers and fire-resistant fabric coverings and in the manufacture of synthetic rubber. It is likely that these chlorinated naphthalenes and diphenyls will be more widely used in the future, unless nontoxic substances are found.

**Pathology.**—Lesions of the skin, caused by chlorinated naphthalenes and diphenyls, will be discussed subsequently. The organ mainly affected by intoxication from the chlorinated naphthalenes and diphenyls is the liver, and in rare instances another organ may be involved, but not to the extent to cause fatalities. After experimenting with rats, Drinker and associates[1] stated that "all the compounds tested attack the liver and the liver alone." Autopsy on human beings reveals acute yellow atrophy and, in one case reported, acute yellow atrophy superimposed upon cirrhosis of the liver. The histologic picture is varied. Beneath the capsule there often extends a wide zone of necrosis; in some sections, efferent veins are surrounded by a single row of well-preserved cells, or by no cells at all. In some areas the liver cells have completely disappeared, and only collapsed stroma remains. In a case reported by Strauss[2] the autopsy findings were as follows: the liver weighed 750 Gm., its surface was smooth, and the capsule showed some shrinking. No subcapsular hemorrhages were present. Parenchyma did not bulge on section. The parenchyma was yellow-brown in color and showed atrophy and degeneration. Microscopic examination of the liver showed universal necrosis of the parenchymal cells with recovery manifest in the biliary ducts, and healing in the stroma.

**Signs and Symptoms.**—Two distinct aspects in the reaction to the chlorinated naphthalenes and diphenyls can be distinguished; namely, the skin effect and the toxic effect upon the liver. The skin lesions are often referred to as "acne," "cable rash," or "blackhead itch." In the medical literature this skin condition is known as chloracne. Small pimples and dark pigmentation of the exposed parts are usually the first signs to be noted. If, however, the exposure is prolonged, when, for instance, the clothing is soiled and the substance penetrates to the skin, the entire body is involved. Comedones and pustules resembling acne vulgaris will then develop. Smith[3] mentions edema as being a typical finding in the patients under her observation, and Strauss[2] noted it in two of his patients.

In those who have suffered systemic intoxication the usual signs and symptoms are jaundice, loss of appetite, nausea, vomiting, and abdominal pain. Jaundice may be an early sign or it may appear only later, being noted often after the patient has been for some time under the doctor's care for other complaints. The time elapsed before systemic evidence of toxicity will present itself varies from a few weeks to many months.

**Laboratory Tests.**—No specific tests have been evolved, but since the liver is the organ in which damage is to be anticipated, the various liver function tests are indicated as confirmatory aids. The deviation from the normal to be expected will be noted in the case histories to be presented. The blood picture will not show signs of a disturbance, unless the illness has been prolonged. The urine may reveal the presence of bile and urobilinogen.

Cotter[4] reports a survey of 2,500 men engaged in coating wire cables for the Navy and summarizes the findings of seven cases in which liver damage occurred. He states that there were deviations in the serum cholesterol, serum phosphatase, serum bilirubin, and serum proteins. The cephalin flocculation tests gave no decisive results until late in the disease.

**Disability.**—If the intoxication does not prove fatal, complete recovery can be expected.

**Treatment.**—Prevention should start with the examination of the worker before he is assigned to a job in the course of which these substances are employed. The examination should establish his physical fitness, and a laboratory investigation of liver function should be a routine pre-employment requisite. Some writers have suggested that a detailed history of possible past liver diseases, jaundice, or syphilis should be carried out to eliminate unfitted applicants from this type of work. But dependence upon this procedure is unreliable, since patients are too often ignorant of their own past systemic disturbances. It is, therefore, suggested that in addition to a rigid pre-employment examination, a similar periodic check-up of all workers should be carried out as a routine procedure.

Adherence to personal hygienic measures should be obligatory, and the workers should be supplied with adequate means of maintaining good hygiene through individual lockers, the one for their working clothes and the other for their street clothes.[5] Showers should also be provided so that, following the removal of their work clothes, workmen can cleanse themselves thoroughly before putting on their street clothes. All work clothes should be laundered every other day. Protective clothing should be provided. In addition, the plant should be kept meticulously clean and all dust or deposits of wax from chlorinated naphthalenes and diphenyls removed during the period between work hours. Engineering control measures should be kept at the highest degree of efficiency and constantly checked for a possible breakdown of protective installations.

In examining a worker who has developed a systemic disturbance, attention should be directed to the liver disorder. This may be treated much as described under tetrachlorethane, the chief points being a high carbohydrate, high protein diet with adequate fluid intake, and particularly intravenous infusions of 5 to 10 per cent glucose in saline in amounts of from 2,000 to 3,000 cc. or more daily. The use of calcium and of sodium xanthine experimentally has been of no value in prevention or treatment of the liver damage due to the chloronaphthalenes. This differs, for example, from the treatment of carbon tetrachloride poisoning in which calcium products are of much value.

The following history was taken from the article by Strauss[2] which has been previously mentioned.

"In December of 1942, six months after exposure to chlorinated naphthalene fumes and three months after development of dermatitis, a white male adult, 50 years of age, became jaundiced.

"His only other symptoms were loss of appetite and weight. He stated that his usual weight was 189 lbs. and he now weighed 173 lbs. There were no complaints of abdominal pain, gastric distress, or change of bowel hygiene.

"On January 22nd, 1943, the plant physician sent him to a local hospital for study.

"The patient appeared to be well developed, a well nourished, male adult, lying quietly in bed, in no apparent distress. There was apparent loss of weight. The skin had an icteric tint. There was evidence of dermatitis of both arms, face, and back of neck.

"The sclera of the eyes was jaundiced, the pupils were equal, regular, and reacted to light and accommodation. The nose was negative. There was evidence of a post-nasal drip in the throat. There was complete edentulation. The heart rate was regular, the sounds were of good quality, with no murmurs, and the size was normal to percussion. The blood pressure was 102/60. The lungs were clear and resonant throughout.

"The abdomen was soft, no masses palpable. The liver was not palpable. There seemed to be some diminution of the size of the liver to percussion. The spleen was not palpable.

"On January 22, the positive urine findings were bile 2 plus, few fine granular casts and occasional pus cells. On January 23, bile 1 plus, many fine granular casts. A blood count revealed 4,730,000 red blood cells, hemoglobin 100 per cent, color index 1.05. Slight anisocytosis and slight poikilocytosis, few macrocytes and rare microcytes. White blood cells 3,800, segmented forms 68 per cent, stabs 5 per cent, lymphocytes 34 per cent, monocytes 1 per cent, eosinophiles 1 per cent and basophiles 1 per cent.

"On February 2nd, the patient was referred by his family physician to a large medical center for further study. He remained in this institution until February 11, 1943. The additional data obtained was reported as follows:

"The history was essentially the same as reported above. The physical examination the same as reported above except that in addition, bilateral, small, indirect inguinal hernias were noted.

"Red blood cells 7,520,000, hemoglobin 106 per cent, white blood cells 6,250, segments 60 per cent, lymphocytes 32 per cent, monocytes 4 per cent, eosinophiles 2 per cent, basophiles 2 per cent. Urea nitrogen 9 mg., bilirubin 11.6, cholesterol 500 mg., serum protein 6.6 per cent, serum albumin 3.8 per cent, serum globulin 2.8 per cent, prothrombin time 19.3 seconds (normal 18-24), cephalin cholesterol flocculation test 2/3/43—2 plus, 2/8/43—3 plus.

"On March 9th, the patient was admitted to a third hospital with the chief complaints of weakness, itchy skin, dyspnea, epigastric pain, loss of appetite and insomnia.

"Physical examination of March 9, 1943, showed the entire body had a deep orange discoloration, conjunctiva had a deep yellow discoloration and also evidence in both eyeballs of conjunctival hemorrhages. The entire body was covered with an elevated puckered dermatitis which was not uniformly spread out about the body, but in most parts, it appeared clustered. The skin rash was especially noticed about the cheeks, chin, neck and both lower and upper extremities. Other areas on the trunk of the body were also covered with the rash; a few of these were bleeding, especially on the trunk and forearms. The tongue was dry and fissured.

"The abdomen was extremely distended with some evidence of free fluid. Liver margin could not be palpated nor could the spleen be palpated. There was no evidence of any masses or tenderness except a slight tenderness in the upper epigastrium on pressure. Remainder of the physical examination was the same as previously reported.

"Impression—'It appeared from the rash as seen on the body and extremities, together with the marked jaundice, and together with the history of exposure to toxic fumes, that an occupational toxic hepatitis must be strongly considered. There was no evidence at any time of any malignancy although this could not be ruled out except by postmortem examination. Acute carcinoma of the pancreas must be considered although there was no evidence of this condition at the time.'

"On March 15, 1943, the red blood count was 5,030,000, hemoglobin 97 per cent, white blood cells 9,300,000, segmented forms 74 per cent, lymphocytes 24 per cent, eosinophiles 1 per cent, monocytes 1 per cent.

"The bleeding time on the same date was 2 minutes, 15 seconds; coagulation time 7 minutes, icteric index 100, van den Bergh, positive immediate direct reaction, cholesterol 384 mgms. (normal 140-180).

"On March 18, 1943, the bile findings were positive. No tumor cells were seen. Specific gravity, 1.010. Direct smear, no organisms found. Cultures were made on March 24, and were sterile after seven days.

"Treatment consisted of high protein diet and intravenous glucose.

"On March 18, 1943, an abdominal paracentesis was performed and 5,000 cc. of orange colored fluid obtained. March 24 the patient went into coma and died on March 25.

"On postmortem examination, the peritoneal cavity contained estimated 2500 cc. of dark orange and lightly colored fluid. The peritoneal surface was smooth and glistening. Beneath the peritoneum of the lateral walls of the abdomen and the diaphragm were extensive, prominent networks of tortuous venules. The deep veins of the abdominal walls were dilated. There were no adhesions.

"Grossly, the esophagus did not show any prominent varices. The stomach contained about 500 cc. of turbid grayish chyme. The pancreas was hard and rather nodular, but lobulation was well defined with occasional pin-head size foci of opaque yellowish material between the lobules.

"The liver weighed 1020 gms. The organ was very small. It measured 21 cm. in width, 16 cm. in length, and 6 cm. in depth through the right lobe. It was generally dull pale yellow in color. The surface was characterized by soft irregular rounded elevations exhibiting prominent lobular markings contrasting with extensive depressed areas, finely granular and wrinkled and without lobular markings. These portions of the liver were rather flabby in consistency and on cut surface contrasted sharply with the nodular elevations above described. The consistency of the liver as a whole was increased. The edge varied from thin and sharp to rounded. The left lobe and spigelian lobe were relatively more reduced in size than the right lobe.

"The diagnosis was cirrhosis of the liver; subacute necrotizing hepatitis (acute yellow atrophy of liver); ascites; fat necrosis of the pancreas; chronic-acneform dermatitis.

"Cause of death was cirrhosis of the liver due to subacute necrotizing hepatitis (acute yellow atrophy of the liver)."

## Naphthalene

Industrial poisoning due to naphthalene is so rare that it will be mentioned only in passing. Naphthalene is widely used in the manufacture of moth repellents and also to a lesser extent in dyes and synthetic resins and in the production of phthalic, maleic, and malic acids. Inhalation may produce headache, nausea, vomiting, and perspiration. It is reported that severe intoxication results in hematuria and edema of the kidneys. By experimental feeding of animals with naphthalene, cataracts of the eyes have been produced. Handling of the solid naphthalene crystals will cause a dermatitis in susceptible individuals.

The only reason for mentioning naphthalene separately from the chlorinated naphthalene is because occasionally a physician will fail to distinguish between naphtha and naphthalene. However, naphtha is an aliphatic hydrocarbon of the petroleum series, whereas naphthalene is an aromatic hydrocarbon, a distillate of coal tar. The following brief case report illustrates the point in question:

In 1943, a 47-year-old male who handled naphtha, kerosene, and gasoline developed failing vision of the right eye. He consulted several physicians and it was finally suggested to him that his occupation was the cause of the trouble. The physician had noted the word "naphtha" in the occupational history of his patient and had confused it with "naphthalene." This erroneous assumption on the part of the physician induced the workman to make a claim to the insurance carrier, resulting in unnecessary litigation.

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# CHAPTER XV

# MISCELLANEOUS ALIPHATIC COMPOUNDS

## Carbon Disulfide

Investigators in almost every industrial country have called attention to the harmful effects of this gas, but in the United States it has received scant notice. As far back as 1865, Delpech*, in France, recognizing its devastating effects upon human tissue, remarked, "He who works in sulfur ($CS_2$) is no longer a man."

**Occupational Hazards.**—Carbon disulfide once found widespread use as a solvent, but because of its deleterious effects it was later largely replaced by less noxious substances. Industry, however, still finds it indispensable in certain processes, the chief of which are these: as a solvent in the extraction of fats, oils, rubber, sulfur, iodine, and the oils from seeds and bones; in the preparation of some lacquers and varnishes; as an insecticide and disinfectant; and finally and most especially in the manufacture of rayon (viscose rayon). It is mistakenly thought that this fast-growing industry is located only in the Pennsylvania-Delaware area. Actually there are fifteen or sixteen plants engaged in the manufacturing of artificial silk scattered elsewhere in the United States.

The average industrial physician rarely comes in contact with illness resulting from chronic carbon disulfide poisoning; the medical literature is devoid of articles concerning it; textbooks of medicine ignore it, and even recent publications dealing with occupational diseases fail to describe it. Until Gordy and Trumper[1] made their report in 1938, no papers dealing with this subject had been published in America since 1905, although Dr. Alice Hamilton had urged an investigation of the rayon industry in 1937.

In 1938, with the cooperation of the United States Department of Labor,[2] the medical staff of the University of Pennsylvania, under Lewy, the American Chemical Society, and the Pennsylvania Department of Labor and Industry, the first coordinated group study of this gas ever to be conducted, either in the United States or in foreign countries, was made. Because of the inexperience of the profession with this substance and because of the fact that a better understanding will become obligatory for all industrial physicians, a résumé of the aforementioned survey is presented.

**Résumé of Group Study.**—Carbon disulfide belongs to the anesthetic group of noxious gases. It is a colorless, transparent fluid which is inflammable and highly explosive, an additional reason why it was largely discarded as a solvent. It enters the body through the lungs and is immediately carried by the blood stream to all the organs, including the brain. It is eliminated through the urine, skin, and lungs. Lehmann[2] concluded, following his experiments, that from 80 to 85 per cent of the inhaled carbon disulfide is again exhaled, leaving from 10 to 15 per cent to be retained. If poor ventilation is present, exhaled fumes may be returned to the workman in the atmosphere to be again inhaled. Thus intoxication is dependent upon the relationship between the intake and the exhalation of $CS_2$. Dissipation of the fumes at the same pace as the intake will prevent the appearance of symptoms.

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* Delpech, A.: Industrie du caoutchouc souffle. Recherches sur l'intoxication speciale qui determine le sulfure de carbone, Annales d'Hyg. publique et de Med. legale, 1863, deuxieme serie, XIX, 65.

Artificial silk, or rayon, is made by a viscose process, which is the converting of cellulose into a gelatinous mass by the addition of sodium hydroxide and carbon disulfide. During this process, hydrogen sulfide is evolved. This by-product is less noxious and damaging than carbon disulfide. Carbon disulfide has a specific gravity of 2.62 as compared with air, and hydrogen sulfide, 1.17 as compared with air. Being heavier than air, both of these substances hang in the breathing zone of the workers. Following their investigation, the Pennsylvania Department of Labor and Industry set the following standard of the permissible limits of $CS_2$ and $H_2S$ in the breathing zone of the workers: 10 p.p.m. for $CS_2$, when existing alone, and 10 p.p.m. for $H_2S$, when alone. If combined, then 5 p.p.m. of each.

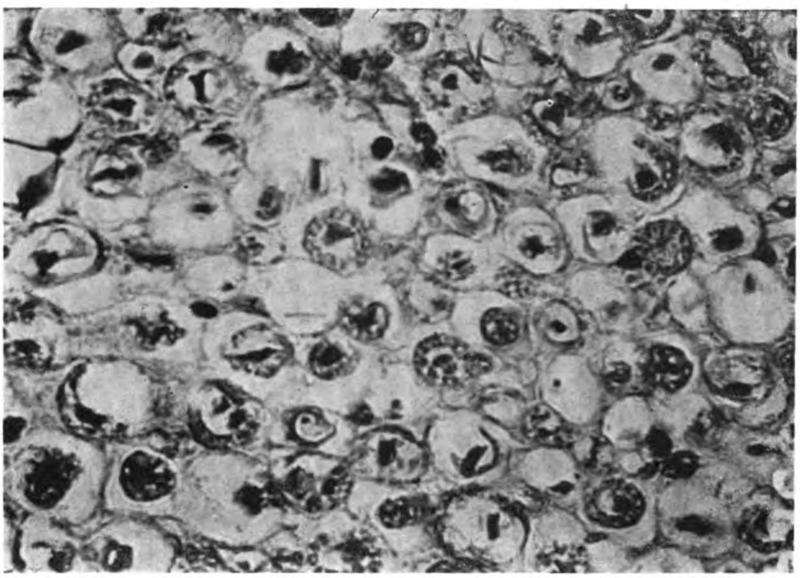

Fig. 7.—Cross section of the sciatic nerve of a dog with experimental $CS_2$ poisoning. The myelin is swollen and fragmented. The axis cylinders are unequal in diameter, some swollen while others are extremely thin. (Lewy.)

**Pathology.**—Primarily this gas produces severe *neural poisoning.* This can be understood when it is realized that $CS_2$ is a strong solvent for fat and that the neural tissue function is dependent upon the lipoid

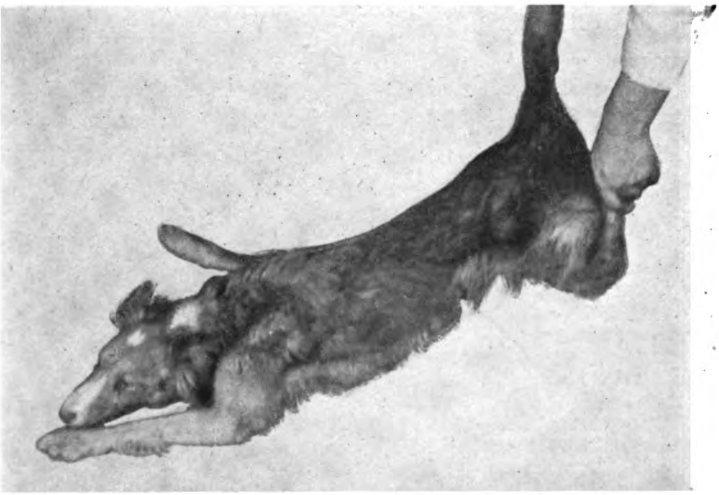

Fig. 8.—Dog 165. After six weeks' exposure to 400 parts per million $CS_2$, eight hours daily, five days a week. The animal is unable to stand on its forelegs with the back legs supported. The dog is in partial flaccid paralysis. (Lewy.)

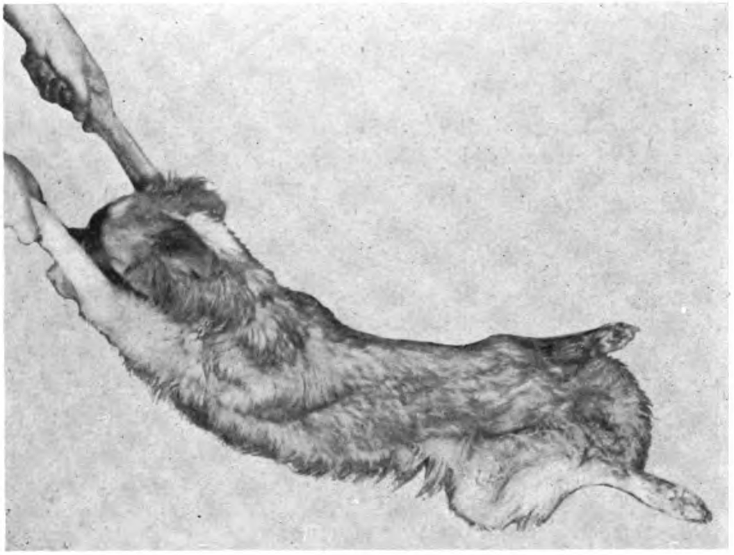

Fig. 9.—Same dog unable to stand on hind legs with the forelegs supported. (Lewy.)

setup in the nerve fibers, myelin sheaths, and cytoplasm (Fig. 7). Any portion of the nervous system may be affected, thus accounting for involvement of the sensory (Figs. 8 and 9), motor, mental, or physical functions. *Blood changes* following disulfide exposure and intoxication are for the most part consistent with a secondary anemia, but no typical picture can be adhered to except for a more or less uniform abnormality

Table VII. Indicated Symptoms of Carbon Disulphide and Sulphuretted Hydrogen Poisoning Totalled From Questionnaires of 159 Viscose Rayon Workers

| | |
|---|---|
| *I. Brain:* | |
| Restlessness | 115 |
| Talkativeness | 27 |
| Easy excitability | 100 |
| Irritability | 120 |
| Change of character | 66 |
| Apathy | 80 |
| Withdrawal | 78 |
| Chronic fatigue | 118 |
| Depression | 82 |
| Listlessness | 34 |
| Difficulty of concentration | 31 |
| Forgetfulness | 37 |
| Partial loss of memory | 29 |
| Headache | 99 |
| Vertigo | 97 |
| Insomnia | 109 |
| Day drowsiness | 99 |
| Hallucinations: sight, hearing | 49 |
| Ideas of persecution | 15 |
| *II. Nerves:* | |
| Hands heavy | 50 |
| Hands numb and tingling | 57 |
| Weakness of muscles: legs, arms | 91 |
| Loss of sensation | 18 |
| Difficulty going upstairs | 65 |
| Muscular pain | 98 |
| *III. Disturbance of Vision:* | |
| Dimness of vision | 65 |
| Cloudiness of vision | 64 |
| Narrowing of field of vision | 7 |
| Change in color perception | 7 |
| Double vision | 12 |
| *IV. Digestive System:* | |
| Loss of appetite | 68 |
| Loss of weight | 76 |
| Indigestion | 92 |
| Pain in stomach | 63 |
| Nausea | 59 |
| Constipation | 91 |
| Diarrhea | 12 |
| Vomiting | 16 |
| Shortness of breath | 34 |
| Pain behind breast bone | 31 |
| Abdominal pain | 21 |
| Palpitation | 23 |
| Ulcer of stomach (reported) | 6 |
| *V. Characteristic Color of Complexion:* | |
| Color of complexion | 38 |
| *VI. Reported Loss of Libido:* | |
| Loss of libido | 50 |
| *VII. Significant Symptoms of Sulphuretted Hydrogen Poisoning:* | |
| Irritation of eyelids | 56 |
| Inflammation of eyelids | 72 |
| Pain in eyes | 54 |
| Feeling of sand in eyes | 68 |
| Photophobia | 61 |
| Tearing or burning of eyes | 31 |
| Haloes around lights | 34 |

of the monocytes. These show a relative and absolute increase in numbers, as well as characteristic alteration in the morphology and staining properties. They contain multiple minute neutrophilic granules and often numerous vacuoles. Creskoff[2] termed them "toxic monocytes" and felt that their presence in the blood picture was indicative of a pathologic state.

### Signs and Symptoms.—

ACUTE INTOXICATION.—Acute intoxication is rare in industry. In the mild to moderately severe cases the picture is that of alcoholic intoxication, with headache, nausea, reeling, giddiness, talkativeness, laughing, crying, or stupor. More severe cases may continue through this initial stage into coma and death.

CHRONIC INTOXICATION.—Table VII is reproduced from the study of 159 viscose rayon workers and taken from the aforementioned Pennsylvania study.

DISTURBANCE OF VISION.—Of this vast and varied symptomatology outlined, one of the most frequent and earliest disturbances noted is altered vision. Objects become indistinct and appear to be veiled in a color fog. Failure of vision is fairly rapid. In a report of this phase of the investigation Robb McDonald stated[2]:

> "The failure of vision or amblyopia is known to be produced by several exogenous toxins, such as lead, carbon disulphide, tobacco, and alcohol. Fortunately, a differential diagnosis between these poisons by means of special examinations as to the determination of the fields of vision, especially the central fields, is usually possible. Carbon disulphide causes a scotomata (a blind area) in the center of the visual field where the vision is most acute. This is called a central scotoma and it has several characteristics that distinguish it from the scotomas due to lead, nicotine, or alcohol. Some of these toxins also cause a contraction of the peripheral field of vision. The loss of vision comes on more rapidly in carbon disulphide intoxication than in any of the other intoxications and also shows more rapid improvement if the source of intoxication is removed.
>
> "These changes in the visual acuity are due to carbon disulphide affecting the optic nerve of the visual cells of the retina. Chronic mild inflammation of the optic nerve may cause an enlargement of the blind spot. (This is a small blind area in the field of vision denoting the area where the optic nerve enters the back of the eye. It is present in every eye and must not be confused with a central scotoma which was mentioned above.) In some affections of the optic nerve, an enlargement of the blind spot precedes the development of a central scotoma. In the observations during the viscose rayon survey, approximately one-half of the workers gave a history of some ocular trouble. However, no one was seen in whom diminution of vision could be objectively proven to be due to a central scotoma. Two gave a history very suggestive of having had a central

scotoma at some previous time and one was found to have poor central color vision.

"Enlargement of the blind spot was found in slightly less than one-quarter of those examined and was found with greater frequency in those employed in the churn room than elsewhere. This phenomenon has not previously been noted in the literature on carbon disulphide intoxication, and may be of significance, when present, in establishing an early diagnosis."

DISTURBANCE OF HEARING.—Batson[2] concluded that the end organ of hearing was not ordinarily disturbed, but the inability to fix attention by those intoxicated resulted in a pseudo loss of hearing.

BLOOD CHEMISTRY.—A study of the blood chemistry of these patients revealed an increased serum cholesterol above the average and a decreased percentage of esterified cholesterol, no matter what the severity of their intoxication. For a variable period after exposure the blood and urine contained a significant and determinable amount of $CS_2$.

COMPOSITE SYMPTOMATOLOGY.—Gordy and Trumper,[3] who deserve much credit for their constant contributions to knowledge of this solvent, augmented their previous reports with twenty-one additional cases. The symptomatology noted by them in these cases is as follows: 90 per cent showed encephalopathic features varying from lethargy, anxiety states, and irritability to vertigo. One of these was frankly psychotic; practically 75 per cent showed diminution or loss of libido; 70 per cent revealed varying degrees of neuropathy; and 67 per cent had subjective eye symptoms; more than two-thirds had gastrointestinal disturbances; more than one-half had varying degrees of asthenia, headache, muscular stiffness, and dyspnea; and a few had a picture simulating angina pectoris; 55 per cent of these victims had symptoms which pointed to localized lesions of the central nervous system, including parkinsonism; 30 per cent had vasomotor symptoms, and a similar percentage showed blurring of the optic disc.

**Differential Diagnosis.**—One should be very reticent about accepting a case as one of carbon disulfide poisoning, because of its rare use. Alleged cases demand immediate contact with the plant or industry to certify its presence or absence in the patient's working conditions.

Among the aids to differential diagnosis of $CS_2$ poisoning, the reaction of corneal and pupillary reflexes ranks first.

CORNEAL REFLEX.—Marked decrease or loss of the corneal reflex, especially if bilateral and not accompanied by sensory disturbances in the distribution of the fifth nerve, is an extremely rare event in diseases other than $CS_2$ poisoning.

PUPILLARY REFLEX.—The same consideration holds true in relation to the pupillary reflex in absence of syphilis, epilepsy, or an intracranial mass lesion.

NEUROPATHY.—Finally, in a polyneuropathy, especially with predilection for the peroneal nerve, $CS_2$ poisoning should be considered when other causes have failed to explain the disease. The combination of polyneuropathy with a Korsakoff psychosis gives a picture almost identical with $CS_2$ poisoning. Strangely enough, alcoholism is rare among viscose rayon workers.

SYPHILIS AND TABES.—Diminution or loss of patellar and pupillary reflexes, ataxia, shooting pain in the legs, and loss of libido may raise the question of differential diagnosis between cerebrospinal syphilis or tabes and $CS_2$ poisoning. However, in uncomplicated $CS_2$ poisoning the Argyll Robertson type of pupillary reaction and the characteristic reactions in blood and spinal fluid are absent, and loss of vibratory or position sense has not been observed.

MULTIPLE SCLEROSIS.—Differentiation between multiple sclerosis and $CS_2$ poisoning, though often impossible in the beginning, is not difficult in a fully developed case. So, also, the differential diagnosis against postencephalitic parkinsonism and senile paralysis agitans is difficult only in the early stages.

VARIOUS ERRONEOUS DIAGNOSES.—It may best serve the purpose of pointing out how to avoid a wrong diagnosis to enumerate some erroneous diagnoses recorded in various hospitals on histories of patients who were actually suffering from chronic $CS_2$ poisoning: *ovarian deficiency* because of loss of menstrual period and vicarious severe headache; *pituitary tumor* because of loss of vision, pallor of discs, loss of libido, temporal headache; *mastoid disease* or *tumor of the auditory nerve* because of ringing in the ears, subjective deafness, and vertigo; *thyroid gland disease* because of tremor, profuse sweating, and loss of weight; *tuberculosis* because of loss of appetite, cough, shortness of breath, and night sweats; *blood disease* because of increased number and pathologic forms of monocytes.

IMPORTANCE OF QUESTIONING PATIENTS.—It may be difficult to diagnose chronic $CS_2$ intoxication if the physician *does not ask* for and the *patient does not mention* the hazards of his occupation. The occurrence of $CS_2$ poisoning and the opportunity for frequent contact with persons suffering from the effect of this poison are obviously linked with a comparatively small number of industrial centers. Many an experienced practitioner and specialist will never actually see a case of $CS_2$ intoxication. Nevertheless, carbon disulfide is now a recognized industrial poison in this country. It is, therefore, important that every physician question his patients about their past and present occupation and keep himself fully acquainted with industrial hazards and occupational diseases. This cannot be overemphasized.

### Medicolegal Aspects.—

TEMPORARY DISABILITY.—Effects from mild exposure tend to disappear soon after removal from the exposure.

PERMANENT DISABILITY.—This is not to be expected in the majority of cases. No conclusions have been drawn regarding the psychiatric stigmas of carbon disulfide intoxication. Concerning the peripheral neuropathies, weakness and paralysis of the muscles return to normal gradually when exposure is no longer encountered. Advanced paralysis may take from nine months to two years for recovery. The expectancy for full recovery from most of the neurologic disturbances is good, although from months to several years may be required. This may also be said for visual damage.

### Treatment.—

PROPHYLAXIS.—The most important point is prevention of such poisoning. Preventive measures consist primarily of rigid examination of the plants, to insure modern, safe equipment and the presence of adequate ventilation. The storage and pipe conveyance of carbon disulfide must be satisfactory, and explosions should be guarded against. Medical examination of those exposed to this hazard should be at intervals not greater than one month. Men should be taught to report the first intimation of any unusual symptom; and if this solvent is the suspected cause of the complaints, the employee should be at once removed from the hazard. The drinking of alcoholic beverages by those likely to be exposed to carbon disulfide should be discouraged.

DIET.—To date, no adequate or specific treatment for cases of chronic carbon disulfide poisoning is known. It would seem likely in view of the similarity of many of these cases to Korsakoff's syndrome, that a diet high in vitamin content with the vitamin B complex as an adjunct would be of value. If vitamin B complex is to be used, the dosage should be relatively large; for example, from 50 to 60 mg. of thiamine chloride parenterally daily; 200 mg. of nicotinic acid daily; riboflavin, 1 mg. three times per day; vitamin $B_6$, 20 mg. twice daily parenterally, and so on. Liver extract in dosages similar to those advised in the chapter on manganese might be tried.

PSYCHIATRIC AND OPHTHALMOLOGIC ATTENTION.—The aid of a psychiatrist will be needed for most of these patients, since a program of mental hygiene will need to be instituted. The ophthalmologist should be consulted on problems related to the eyes.

Exercises for existing muscular weakness, sedation, and tonics may be of value. If parkinsonian-like symptoms are present, a trial of those drugs mentioned in the chapter on mercury for control of these manifestations might be made.

The *acute case* will rarely be seen. When such a case does occur, artificial respiration, from 5 to 7 per cent carbon dioxide in oxygen inhalations, and the respiratory and circulatory stimulants mentioned in the chapter on petroleum hydrocarbons are used.

**Case History.**—This case was reported by Gordy and Trumper.[1]

"*Present Illness.*—A man, aged forty-nine, foreman in a viscose factory who worked principally in the reeling room, first became ill in April, 1935. Headache, vertigo, rapid loss of weight, and paresthesia in his hands and feet developed. With this there was also insomnia, causeless weeping, and night terrors. He had had aching in his bones for months. For some time he felt that his mind was not working properly. Both his wife and his son had noticed that his actions had not been normal and had told him about his crying and screaming while sleeping and that his body shook all over. His memory was unreliable. He described episodes resembling fugues in which he would find himself at some place and could not remember how he got there. He was unable to drive a car because he was uncertain of himself. He complained of dyspnea and a sense of oppression in the chest. He also had episodes of blindness lasting for a few minutes and also double vision. He was unable to retain a position because of his memory faults. He had a marked diminution of libido.

"*Examination.*—The patient was fairly well built and gave evidence of having lost weight, his weight at that time being 156 pounds (71 kg.). The pupils were regular and reacted to light and to accommodation. The eyegrounds showed slight pallor of the temporal discs and slight atherosclerosis. The cranial nerves were normal except for mild tremor of the tongue. No ataxia or other evidence of cerebellar dysfunction was present. The biceps, triceps, knee, and ankle jerks were present and equal; there were no pathological reflexes and no disturbances in any of the modalities of sensation.

"*Blood pressure,* 150 systolic; 100 diastolic.

"*Mental Status.*—The patient tended to be morose, apathetic, somewhat indifferent, and his thought processes were retarded. He was oriented in all spheres, but his memory was definitely dulled. He found it difficult to place various occurrences in the past in any sort of proper sequence. He had formerly had the reputation of being a first-class worker, with an ability to handle other employees. At the time of examination he had a marked lack of initiative and a sort of childlike dependence on others."

## SULFUR COMPOUNDS

### Hydrogen Sulfide

Hydrogen sulfide in a concentrated form is deadly and, like hydrocyanic acid, exceedingly rapid in its action. Its characteristic odor, like that of rotten eggs, is familiar to all. It is a transparent gas, readily soluble in water, and is the natural product of putrefaction of animal or vegetable matter. Because of its intense odor it is easily recognized even in minute amounts.

**Occupational Hazards.**—This gas is never used directly in industry but is formed frequently as a by-product of certain processes. It is to be found in sulfur dye works, tanneries, in the production of carbon disulfide, and in the heating of certain rubbers containing sulfur compounds and may offer a hazard to those working in sewers, mines, wells, caissons, and tunnels. As reported under carbon disulfide, it exists in the process used for the manufacturing of artificial silk or rayon. It is invariably present to some degree in the refining of oil.

**Pathology.**—This gas is both an asphyxiant and an irritant. When death results, it is from respiratory failure and occurs within a few minutes, although the heart may beat for a short period after respiration has ceased. It is important that those first on the scene of action remember this and institute artificial respiration. Persons seemingly dead have been resuscitated by such means. Locally it is an irritant; systemically it affects the nervous system. This may range from drowsiness or sleepiness to convulsions, delirium, deep coma, or death. Because of its action upon the vagus nerve, the heart action is slowed. Within the blood stream hydrogen sulfide is rapidly oxidized without any toxic products resulting from this oxidation. The pathology resulting from poisoning by this gas is apparently transient, for if death does not occur because of asphyxiation, recovery is usually complete. It is significant that at post-mortem examination in those cases where death has been instantaneous the tissues reveal nothing characteristic.

**Signs and Symptoms.**—These can best be described by listing the reactions as they occur at certain concentrations. At a low concentration of between *100 and 200 p.p.m.* there exists a mild irritation, especially to the eyes. This usually results at the end of a day's exposure. Within one-half to one hour *500 p.p.m.* will bring about excitement, headache, dizziness and staggering, diarrhea and dysuria, to be followed sometimes by bronchitis or bronchopneumonia. Short exposures of fifteen minutes to concentrations of from *800 to 1,000 p.p.m.* intensify the previously mentioned symptoms or may result in death. Above this may mean instant death.

CHRONIC POISONING.—Chronic poisoning results in headache, inflammation of the eyes or eyelids, digestive disturbances, loss of weight, and general debility. This constant exposure is confined usually to chemists or testers in oil refineries.

**Medicolegal Aspects.**—

TEMPORARY DISABILITY.—This may be from a few minutes to a few hours or a few days. If a pulmonary complication arises, the disability is for the course of that affection.

PERMANENT DISABILITY.—This is rare, if ever. As in cyanide poisoning, if the dose is not fatal, complete recovery occurs.

**Treatment.**—If this substance is likely to be present in an industrial process, *masks* should be worn which would not only prevent inhalation of the gas but also protect the eyes against irritation. *Ventilation* should be adequate.

COMBATING RESPIRATORY FAILURE.—In severe poisoning, chief attention is directed to the respiratory failure, which appears to be due to depression of the respiratory center. The patient should at once be moved from the point of contact with the gas and fresh air provided. Artificial respiration is started at once, and as soon as available inhalations of mixtures of from 5 to 7 per cent carbon dioxide, with oxygen, are commenced. The use of respiratory stimulants—such as caffeine sodium benzoate, 7½ grains (0.5 Gm.); coramine, 0.5 cc. intramuscularly or preferably intravenously; camphor in oil, from 1 to 2 cc., intramuscularly; and metrazol, 1½ grains (0.1 Gm.)—may be of value here because of the central character of the respiratory failure. Bronchitis and bronchopneumonia may appear as complications and require the ordinary treatments for these conditions.

CARE OF EYES.—The eyes may be severely irritated, and the condition may even cause the development of corneal ulcers. As with other irritants to the eyes, for example, ammonia, they should at once be thoroughly washed with water. Following this the use of a saturated boric acid solution and of olive oil is advised. Local anesthetic solutions, such as 2 per cent butyn or 0.5 per cent pontocaine hydrochloride, may be used in relief of pain. Continuous warm boric compresses may be of value in preventing further complications. When available, the advice of an ophthalmologist should be sought.

CASE 1.—While working for an oil company in the Long Beach refineries, a cracker stillman's helper started out at 6:30 A.M. to make his rounds at gauging the tanks. Failing to return, he was found one-half hour later lying in six or seven inches of water, face down. Medical aid did not reach him until one-half hour later. The medical examination at that time revealed a man of apparently 26 or 27 years of age. No cardiac sounds could be heard; the pupils were dilated and fixed. He was deeply cyanotic. The extremities were relaxed and moderately warm. There was a minor abrasion about ⅜ inch in diameter over the left temple, with no swelling. The neck did not appear to be out of line, and no crepitation could be heard upon manipulation. He failed to respond to cardiac stimulants, and the pulmotor squad using carbon dioxide-oxygen inhalations obtained no results. The autopsy examination showed water in the lungs; otherwise it was entirely negative. No injury to the brain or body was found, and the heart was normal.

The top of the tank which this man was gauging was covered with a half foot of water to prevent the escape of gases. At three places around this tank there were escape valves or pipes, and at one place there was a covered gauging hole. This hole is covered by a flap top, which is opened by a mechanical foot pedal. It is so situated and arranged that usually the wind blows the escaping gas away from the workman. On this particular morning the wind was toward the position the workman would have to assume in operating this gauge. It was, therefore, felt that when the victim lifted the lid he received an unusually high concentration of hydrogen sulfide, was overcome, and fell forward into the water.

In this particular refining process, they were engaged in "cracking" the oil into different fractions. This was being done by heating this oil to a temperature of a 1,000° C. At this point the hydrogen sulfide given off is abundant and highly concentrated. The author questioned several petroleum engineers and was told that this concentration is also increased under certain weather conditions.

In the absence of any other obvious cause of death or circumstances which could account for his death, it was held as compensable. The coroner stated that death had been too sudden for any changes to occur that would aid in the post-mortem diagnosis.

Case 2.—A workman engaged in repairing a tank from which hydrogen sulfide was escaping first became dizzy, noticed a pressure in his head, and then became unconscious. He returned to consciousness in eight or ten minutes and was removed to the hospital. There the examination revealed no cyanosis or dyspnea. The pulse was 60 per minute, and the blood pressure 124-78/70. A few scattered moist râles were heard throughout the lung fields. He recovered completely within thirty-six hours and returned to work at the end of four days without any further disability.

## Sulfur Dioxide

Sulfur dioxide is an irritating gas arising from the combustion of sulfur. It is a common component of the atmosphere of industrial communities and can easily be detected in the air. Its concentration is higher in cities subjected to fog. In industry, the maximum amount of concentration allowable for a short time (thirty minutes to an hour) is from 50 to 100 parts of $SO_2$ per million parts of air; for prolonged exposure, 10 parts per million of air.

**Occupational Exposure.**—Its most frequent use is in the operation of small, home refrigeration units. Exposure may also be found in the occupations of sulfuric acid makers, smelters, foundry workers, blast furnace operators, bleachers, cellulose workers, workers at coke ovens, disinfectant workers, dyemakers, petroleum refiners, and vulcanizers.

**Signs and Symptoms.**—Irritation and inflammation to the eyes, nose, throat and lungs, and occasionally digestive disturbances. It is so irritating to the eyes and throat that man usually withdraws from an exposed atmosphere before further damage is done; but instances where withdrawal was impossible are reported, showing edema of the lungs as well as paralysis of the respiratory center. Chronic exposure produces a chronic catarrh, a nasopharyngitis and disturbance of the sense of smell or taste, increased lassitude, and fatigue. Other observers have noticed a lessened tendency to colds, bronchitis, or other respiratory infections. Direct contact of the eyeballs with this liquid has caused frozen eyeballs.

**Medicolegal Aspects.**—

Temporary Disability.—Usually there is a disability of not more than a few days.

Permanent Disability.—There is no permanent disability. Exposure does not induce cardiac disease, tuberculosis, or chronic bronchial disturbances. Removal from the exposure results in a disappearance of symptoms.

**Treatment.—**

IRRITATION OF EYES.—Workers in industrial plants, firemen, or refrigerator repairmen, who are likely to encounter this gas, should wear *gas masks.* Treatment of an eye injured by this gas is similar to that of the eye injured by ammonia. Copious laving of the eye with water, followed by the introduction of saturated solution of boric acid and by the use of a local anesthetic, such as 0.5 per cent pontocaine hydrochloride, is indicated. These are followed by the application of olive oil or some similar oil. Corneal ulceration should be carefully watched for and treated appropriately if it occurs.

RESPIRATORY DISTURBANCES.—For those overcome by the gas, inhalation of from 5 to 7 per cent carbon dioxide in oxygen is used over a long or short period, depending on the severity of the case and the individual patient's reaction. If pulmonary edema is present, oxygen alone is used. In very severe cases, artificial respiration is employed coincidentally with the inhalation. In some cases, the use of respiratory stimulants such as coramine, metrazol, and caffeine sodium benzoate may be of value. A few days of absolute bed rest should be advised for those who have a fairly severe exposure, and this should be continued until all evidence of respiratory embarrassment ceases. During this period codeine phosphate or sulfate, from ½ to 1 grain (0.0325 to 0.065 Gm.), every four to six hours, will alleviate the cough and may frequently be combined to advantage with a soothing cough-syrup mixture. Bland oils should be applied to the exposed membranes. For those who display symptoms of acute asthma, the subcutaneous administration of ½ to 1 cc. of 1:1000 solution of adrenalin may relieve the symptoms. The use of a nebulizer for inhalation of 1:100 adrenalin might also be of value.

**Illustrative Cases.**—The cases seen at our clinic have invariably been very mild. There were no fatalities. In most every instance it was not necessary for the patient to withdraw from his work. The following incident is an example of certain symptoms which may present themselves.

CASE 1.—A 28-year-old male was seen on April 9, 1940. He had previously been seen in February of the same year for a slight exposure to this gas. On the day of this most recent exposure, he and a helper had gone to a home to recharge an electric refrigerator and had taken with them a drum filled with sulfur dioxide. The patient stated that these drums are filled at the plant when cold, and occasionally more is put into them than they can stand. As they were about to recharge the refrigerator, the drum exploded, striking the helper on the lower leg and fracturing it. This occurred in a small, enclosed room, and both workers were almost overcome. The clothing of both was frozen stiff. The older mechanic managed to get his helper outside, where he removed most of the clothing and then proceeded to the hospital.

Examination of the helper revealed the fractured leg (tibia) and a few râles within the lung fields. His chief complaint was referable to the right eye, which had been frozen. He was placed under the care of the ophthalmologist for treatment of the eye and the orthopedic surgeon for the fractured leg. He required no particular medical treatment. The eye slowly returned to normal in four weeks, and the fracture healed without complications. There was a temporary disability of thirteen weeks. There was no permanent disability.

The mechanic complained of pain in the throat and chest and a feeling of constriction. His respirations were embarrassed. Examination showed the throat to be red and the eyes injected; the lungs were full of scattered musical râles, best heard at the beginning of expiration. His picture was typical of bronchial asthma. He was hospitalized for twenty-four hours and given two hypodermics of adrenalin, and thirty hours after exposure the lungs were clear. There were not abnormalities relative to the heart or blood pressure. The temperature was 99° F. on admission and returned to normal by the time of discharge. He remained away from work four days. He was re-examined two weeks later, at which time he was entirely normal. However, he decided to quit this type of work, because of fear of a repetition of his recent experience. The temporary disability in this instance was four days, with no permanent disability.

## References

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2. Group Study: Survey of the Carbon Disulphide and Hydrogen Sulphide Hazards in the Viscose Rayon Industry, Bull. 46, Dept. of Labor and Industry, Pennsylvania, 1938.
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# CHAPTER XVI

# HALOGENS AND HALOGEN COMPOUNDS

## Chlorine

Chlorine (Cl), as well as bromine, is usually credited with producing more serious effects following an ordinary exposure than is substantiated by the facts. Bromine is similar to chlorine in its irritating action. It will, therefore, not be given further mention. Chlorine is a greenish-yellow gas, produced largely from common salt by the electrolytic process, and is sold commercially in cylinders.

It is most frequently used in the bleaching industry; also in the manufacturing of nitric acid, in the dye industry, in textile works, in the making of disinfectants, and in the preparation of various chloride compounds.

**Signs and Symptoms.**—Because chlorine is an extremely irritating gas, an exposed individual usually withdraws before severe gassing can take place. In such instances, irritation to the eyes, nose, and throat will have taken place. Coughing will almost immediately result. In severe gassing the feeling of suffocation will be experienced in addition to the aforementioned signs of irritation. If the lungs are involved, as evidenced by râles, pneumonitis or pulmonary edema may ensue. Pneumonia is a rare sequel, and to be attributable to the effects of chlorine, it should have its onset within a few days following exposure. It is believed that exposure to chlorine does not induce the appearance of pulmonary tuberculosis.

The temporary disability is short, lasting a few days to a couple of weeks at the most, in mild to moderately severe cases. The author has never seen a case of permanent disability due to chlorine gassing, nor has he seen any chronic chlorine poisoning.

**Treatment.—**

IMMEDIATE MEASURES.—The patient severely gassed with chlorine should be removed from the toxic atmosphere promptly, and all constricting clothing about the neck should be loosened. He should be kept quiet in a recumbent position and should be wrapped with warm blankets while hot-water bottles are applied. Oxygen should be administered in all cases. It is considered best not to wait for cyanosis to develop, since the administration of oxygen will often prevent cyanosis and relieve the pain of deep inspiratory effort. Venesection of from 400 to 600 cc. is an extremely valuable measure and should be performed early on all patients who have been exposed to heavy concentrations; but, as already indicated, such exposures in industry will be very rare. The early use of such phlebotomy often prevents development of edema of the lungs, and if used later often relieves pulmonary edema and cardiac embarrassment. The early intravenous administration of 10 per cent dextrose in physiologic solution of sodium chloride may be combined with venesection to combat shock and reduce viscosity of the blood. As with phosgene poisoning, venesection is contraindicated if the gray stage of anoxemia with pallor, collapse, and rapid, thready pulse is reached.

RELIEF OF PAIN AND EXCITEMENT.—Morphine sulfate is not to be used if pulmonary edema and respiratory embarrassment have ensued. Some advise, however, its use for relief of pain and excitement soon after injury. Atropine, epinephrine, digitalis, and strychnine are not of much benefit. Caffeine sodium benzoate, 7½ grains (0.5 Gm.) or from 1 to 2 cc. of camphor in oil given intramuscularly sometimes produces beneficial results.

COMPLICATIONS.—If pneumonia is a complication, oxygen therapy is continued. If a specific infection superimposed upon the injured lung is demonstrated, the use of the appropriate chemotherapeutic agents, for example, sulfanilamide, sulfapyridine, or sulfathiazole, or penicillin serums is advised.

Olive oil in the eyes may be effective. Codeine phosphate or sulfate, ½ to 1 grain (0.0325 to 0.065 Gm.), for cough, or spraying of the nose and throat with a soothing solution, is at times useful, but for this latter use products containing oil should be avoided because of the danger of developing a lipoid pneumonia. The patient should be placated regarding any permanent effects to the lungs. A neurosis may be avoided by the initial attitude of the physician and malingering obviated by the emphatic imparting of existing statistics and knowledge regarding these cases.

## Fluorine

Fluorine (F) is widely distributed in nature, being found in the drinking water as well as in the mineral and soil of many areas of this country. Its chief components are sodium fluoride and hydrogen fluoride. Acute toxic effects attributed to fluorine are due to hydrogen fluoride.

Art glass workers, brewers, bleachers, dyers, aluminum extractors, miners of cryolite and rock phosphate, and magnesium casters may incur stigmas caused by fluorine.

Chronic fluorine intoxication produces dental defects, loss of weight, cachexia, anorexia, and impairment of growth in the young. The most striking finding is that of osteosclerosis, often called fluorosis, which can be detected by x-ray. It has been reported that a relative eosinophilia and lymphocytosis is frequently observed in men who have been exposed for long periods, and several investigators have noted the presence of bronchial asthma in fluorine workers. Burns of the skin will be mentioned in the discussion of hydrogen fluoride.

Machle, Scott, and Largent[1] studied a group of workers who had been intermittently exposed during five years. Analytical determinations showed that the exposure of these workers resulted in absorption of more than three times the normal amounts of fluorine. However, no clinical evidence of any intoxication was demonstrable.

### Hydrogen Chloride

Hydrochloric acid (HCl) or hydrogen chloride, commercially known as muriatic acid, is used by etchers, engravers, metal picklers, and acid dippers and in the electroplating process. It is also widely employed to wash out iron scale, or sludge, from tanks.

Mild exposure causes irritation to the upper respiratory tract. More severe exposure will result in spasms of the larynx, in pulmonary edema, or in pneumonitis of chemical origin. The toxicity of hydrochloric acid in the form of droplets is less than that of anhydrous hydrogen chloride, since the droplets cease to possess dehydrating action. The maximum concentration tolerable for a period of one hour is 50 to 100 parts of hydrogen chloride per million parts of air, while 1,000 to 2,000 p.p.m. is dangerous even in short exposures.

### Hydrogen Fluoride

Hydrogen (HF), or hydrofluoric acid, is a colorless liquid which is easily soluble in water and forms dense white fumes in the moist air, as does hydrogen chloride.

Hydrogen fluoride has extensive uses in the glass industry, in making opalescent electric light bulbs or fluorescent tubes for "neon" signs, as well as in glass etching in the ceramic industry, in the manufacture of fertilizers, in the electrolytic production of aluminum, in certain catalytic processes employed by the petroleum industry, and in magnesium foundries.

In recent years considerable interest has been aroused regarding the action of fluorine and hydrogen fluorine on the lungs. Developments in the fluorescent light industry as well as in certain industries where beryllium oxide is extracted from its ore have served to draw critical attention to the harm caused by fluoride or oxyfluoride. The author prefers to discuss this subject in the chapters devoted to beryllium and to pneumoconiosis.

Hydrogen fluoride has a marked effect upon the skin and underlying tissue. On the skin it causes chemical burns, while subcutaneously its destructive action affects the tissue and blood vessels. In addition to the preventive measures regarding general exposure developed by safety engineers, Benson[2] recommends complete body protection by means of neoprene clothing, including cap, face shield, gloves, neoprene-soled shoes, etc. The following treatment of casualties is recommended by Benson:

"Prompt action is of the greatest importance for successful treatment. For example, when acid has come into contact with the skin, the area affected should be immediately flushed with water for at least ten minutes. The burning sensation of extremely low concentrations may not become apparent for some time after contact, particularly if the acid has touched the tough, thick skin of the hands or feet; therefore, the area should be washed even though there is only a suspicion of an acid contact. High concentrations of acid penetrate the skin rapidly and produce a stinging sensation almost immediately. The warning is sufficient to cause the worker to lose no time in starting first-aid treatment. A delayed-action burn may cause a painful, deep-seated injury that requires medical aid; therefore, splashes of all kinds should be treated for acid burns.

"The following first-aid treatment is recommended:

"1. Flush well with water for at least 10 minutes.

"2. Apply, with cotton, a 10 per cent solution of ammonia (10 per cent of 28 per cent aqueous ammonia solution), and rinse with water. Do not use household ammonia unless none other is available. It is preferable to use a sterile solution made by a pharmacist in accordance with the U.S.P.

"3. Rub well into the affected area the special magnesium oxide-glycerin HF burn salve which should be on hand in the first-aid and emergency booths. Blisters, if any, should be opened with a sterile instrument by a competent person.

"With most acids, merely washing with plenty of water and neutralizing, if done quickly enough, are sufficient to stop their action. HF, however, has a double action on the skin: first, there is the acid, which must be neutralized; second, there is the fluorine ion, which must be precipitated. Therefore, it is necessary not only to wash the area thoroughly and apply ammonia, but also to use magnesium or calcium in a form such that it will precipitate the fluorine ion. If there is delay in treatment, or negect, HF penetrates deeply into the tissues and causes deep-seated burns, which have been compared by one authority to those produced by high-voltage electricity. Blisters are opened so that the salve will reach the acid underneath and stop its action quicker. If the acid has penetrated into the tissue, the magnesium oxide paste applications will not be sufficient to prevent a bad burn, and it will be necessary to obtain medical aid for the injection of 10 per cent calcium gluconate of 20 per cent magnesium sulfate by hypodermic around and under the affected area. This application will form a calcium or magnesium barrier around the burn and prevent further destruction.

"Some of the most painful burns experienced have been under the fingernails. Some physiologists believe that the acid penetrates the nail itself, and when under the nail, starts its destruction of tissue in a particularly tender spot. If the calcium gluconate or magnesium sulfate can be injected through the nail or around it, the pain may be alleviated.

"The above treatment is not the only one advocated by manufacturers of hydrogen fluoride. A number of authorities favor prolonged washing with water to draw the acid from the tissue by osmotic action. One worker with HF recommends prolonged washing followed by ice-cold alcohol immersion. Even the most painful burn can be rendered almost painless by holding the affected part in ice-water; but when it is withdrawn from the ice-water, the pain may commence again.

"It is almost impossible for anyone consciously to inhale enough hydrogen fluoride to harm himself seriously; the acid gas is too pungent for voluntary inhalation. A study of the available literature on this subject showed only one case of a fatality from HF inhalation. The possibility of being rendered unconscious and becoming injured appears to exist only if a man is trapped where gases are suddenly released. Should this occur, the following procedure is recommended:

"1. Remove the patient to fresh air, free from drafts.

"2. Treat skin burns by the prescribed treatment.

"3. Make patient rest.

"4. Reassure the patient and discourage him from coughing. Cough syrup should be provided in each shower house.

"5. Apply artificial respiration if necessary.

"6. Keep the patient warm, because he will be suffering from shock.

"Eye injuries among workers handling HF have been rare; strict adherence to eye protection is apparently the reason. Nevertheless, bubbler fountains should be installed in each shower house for flushing and washing the eyes in case of injury. Bottles containing a saline solution should also be available for neutralization."

## Case Report.—

CASE 1.—A patient was admitted to the service of Dr. Harry Alexander of our clinic with a history of having fallen while carrying a small bottle of hydrogen chloride. The bottle broke, splashing the patient on the left thigh and left arm; the fingers of the left hand were soaked. The immediate treatment administered at the plant had been superficial. He was not brought to the clinic until one-half hour after the accident, at which time the fingers were blanched. Within two hours the distal portions of these fingers were dusky red. The following morning they appeared gangrenous. Subsequently these distal portions had to be amputated.

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2. Benson, R.: Hydrogen Fluoride Exposure, Indust. Med. **13:** 113, 1944.

# CHAPTER XVII

# THE AROMATIC HYDROCARBONS

## Benzol

Benzene ($C_6H_6$) is a coal tar product which should always be referred to as *benzol* in order to distinguish it from the petroleum *benzine* of the aliphatic hydrocarbons. It is a colorless liquid with a not unpleasant odor in low concentrations which, however, becomes disagreeable as the concentration is increased. It boils at 80°C., solidifies at 5.5°C., and is miscible with most of the common industrial solvents.

While the use of benzol in industry has been considerably reduced in recent years, the incidence of benzol poisoning is still fairly frequent. Too often is benzol hidden under a trade name or is carelessly substituted for less toxic solvents.

**Occupational Exposure.**—Benzol is such an excellently efficient solvent that it can be used in any process demanding solvent action. Sappington[1] lists over one hundred occupations in which the workers are likely to be exposed to benzol. As intimated in Chapter VI, it is a fact that a physician will not always find it easy to learn that the patient had been exposed to benzol; he will often have to ferret out such information for himself.

**Pathology.**—From the point of view of interest in pathologic changes, acute benzol intoxication is of little concern. It acts as a depressant on the central nervous system and if it is not fatal, recovery without sequelae is the rule.

Chronic benzol poisoning usually produces severe degrees of injury to the blood-forming organs, and often proves fatal. The blood picture is bizarre and inconstant.

BONE MARROW REACTION.—Before considering the hematologic phase, some attention should be given to the actual pathology. The most marked lesions occur in the bone marrow. For years it has been assumed that a medullary aplasia occurred. Cases are on record in which patients with a known exposure to benzol have been denied compensation because they demonstrated hyperplastic marrow, which was considered not typical of benzol poisoning. Some years ago Hamilton, realizing the variations in the pathology of all blood diseases, predicted that further study would alter the viewpoint then held. Her prophecy has been substantiated in the work of Mallory, Gall, and Briskley,[2] Thompson, Richter, and Edsall,[3] and Rhoades and Miller.[4]

Mallory and co-workers have studied nineteen cases, fourteen of which were completely autopsied, and histologic material was obtained from each of the nineteen. In the autopsied cases, material was obtained from several specimens of bone marrow, including the long and flat bones, as well as from the thoracic and abdominal viscera, liver, and spleen. None of these patients had been exposed for less than six months, four being exposed for less than a year; eight, from one to four years; and seven, from four to twelve years. Excluding two cases of possible leucemia, the remaining number could be divided into two groups, six showing marrows less cellular than normal and nine revealing hyperplasia. In comparing their findings with the history of each case, Mallory and associates were of the opinion that intensity of exposure was not a determining factor in the bone marrow reaction, but that duration of exposure was. Cases of less than a year's exposure showed a *hypoplasia,* whereas those with prolonged exposure evidenced a *hyperplasia,* indicating that the initial effects of benzene upon the bone marrow were to depress it but that the prolonged effect was to stimulate it.

On the other hand, from these observations it might be reasoned that since persons react differently to benzene exposure, the occurrence of a hyperplasia enables the worker to continue for many years, whereas the development of an aplasia results in rapid disability or fatality unless the patient is removed from the exposure.

EFFECT ON THE LIVER.—The chief findings in the liver were the prominence of the Kupffer cells, which were loaded with hemosiderin, and central necrosis, possibly due to terminal infection rather than to the benzene assault. In Mallory's fourteen autopsied cases, the spleen was rarely noted to be enlarged clinically but was frequently found enlarged at the autopsy table. Increased cellular activity was usually present. Well-defined foci of hematopoiesis were seen even in cases of minimal general changes. These became more marked and numerous with increasing severity of the lesion. These investigators noted a general parallelism between bone marrow and splenic hematopoietic activity. In spite of frequent purpura of the skin, mucous membranes, and serous surfaces, they noted no gross brain changes except in one case showing several subarachnoid hemorrhages.

Some evidence seems to exist to the fact that an anemia due to benzol poisoning may become slowly progressive. Markland,[5] in his study of a case of chronic benzol poisoning, noted bone marrow changes typical of pernicious anemia, and in another case the picture suggested myeloid leucemia. Mallory and associates[2] observed two cases of leucemia in their series, and Erf and Rhoades[6] noted such a picture in one of their cases. The histopathologic changes encountered in benzol poisoning are illustrated in Figs. 10 to 28.

Regarding the neoplastic changes noted in their study, Mallory and co-workers[2] stated: "Also suggestive to anyone who has studied even superficially cases of the hyperplastic type, are the evidences of what

Fig. 10.

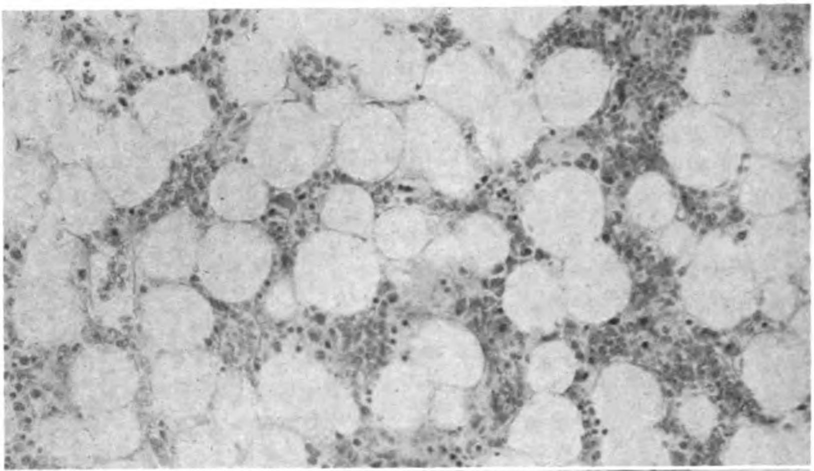


Fig. 11.

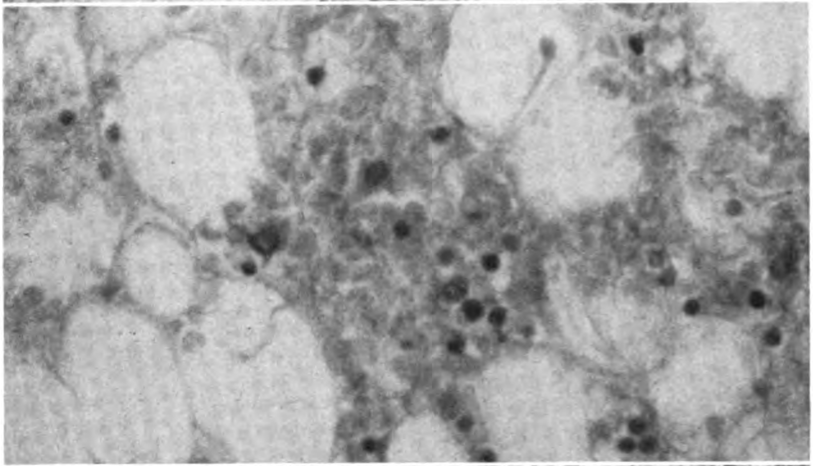
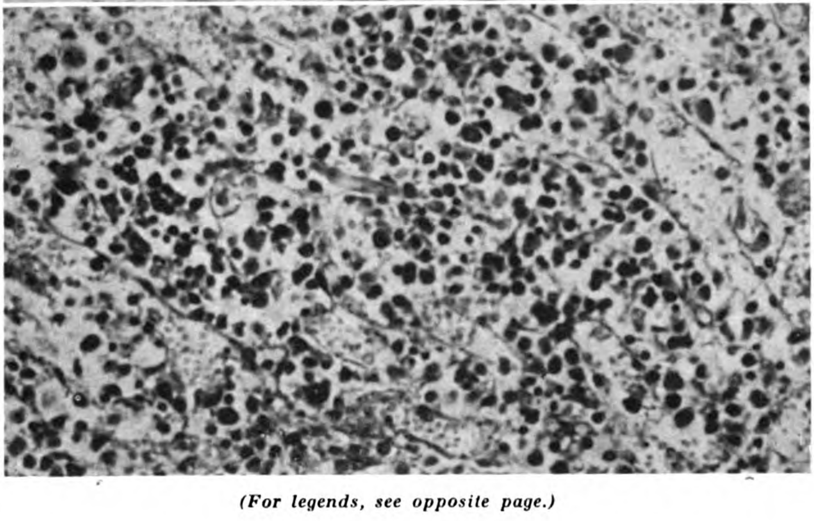


Fig. 12.



*(For legends, see opposite page.)*

for want of a better term may be called a neoplastic tendency. The degree of anaplasia, the rapidity of growth as judged by the number of mitotic figures, the development of cells having no counterpart in normal tissues but common to a variety of malignant tumors are phenomena characteristic of neoplasia which the authors have never met heretofore in such a marked degree in non-neoplastic states."

**Blood Picture.**—It was once believed that the earliest evidence of the effect of benzol exposure is leucopenia. Largely this has been the only test relied upon, and in many plants a routine white cell count constitutes the basis for permitting the workman to continue his work or removing him from the exposure. From the recent investigations such a practice is questionable and probably unreliable, yet textbooks of general medicine continue to cite leucopenia as the characteristic finding in benzol poisoning.

Investigations by Greenburg, Mayers, Goldwater, and Smith,[7] of the New York Department of Labor, and Erf and Rhoades,[6] of the Rockefeller Institute, indicate that leucopenia is not a prevalent isolated finding. Greenburg and co-workers found a deviation of the white count in only 14.5 per cent of 332 workers examined. Of his patients who were daily exposed to benzene vapors, 85 per cent showed a normal white cell count. Furthermore, there appears no basis for assuming that leucopenia is produced in the presence of a severe anemia and purpura.

MACROCYTOSIS.—What appears to be a valuable contribution to the study of benzene not noted in other investigations is the finding of Greenburg and co-workers[7] regarding the mean corpuscular volume, mean corpuscular hemoglobin, and the mean corpuscular hemoglobin concentration. In their series of cases they found a distinct tendency toward macrocytosis. The average cell size was greater than 94 cubic microns in 9.3 per cent of the control group, as compared with 46.7 per cent over 94 cubic microns among those exposed to benzene. The average size was less than 90 cubic microns in 73.4 per cent of the control group, as compared with 30.1 per cent of the group exposed to benzene. They had fifty-five cases in one of the plants inspected which showed a white cell count above 5,000. Upon the basis that benzene causes leucopenia, these cases might have been classified as negative for benzene poisoning.

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Fig. 10.—Bone marrow (vertebral): There is pronounced hypoplasia with replacement of marrow substance by fat. Most of the sinusoids are collapsed. Those which remain patent contain only small clusters of hematopoietic cells, the predominant elements being non-nucleated erythrocytes. (×200.) (Mallory: J. Indust. Hyg. and Toxicol. **21**: [Oct.], 1939.)

Fig. 11.—Bone marrow (vertebral): A higher power view from the same section as Fig. 1. A group of sinusoids distended with erythrocytes is seen. Comparatively few nucleated elements are evident, and these are almost all normoblasts. (×500.) (Mallory: J. Indust. Hyg. and Toxicol. **21**: [Oct.], 1939.)

Fig. 12.—Bone marrow (vertebral): The sinusoids are all widely dilated and filled with hematopoietic elements with normoblasts, erythroblasts, and stem cells predominating. Only a few giant cells are seen. The marrow fat has completely disappeared. (×300.) (Mallory: J. Indust. Hyg. and Toxicol. **21**: [Oct.], 1939.)

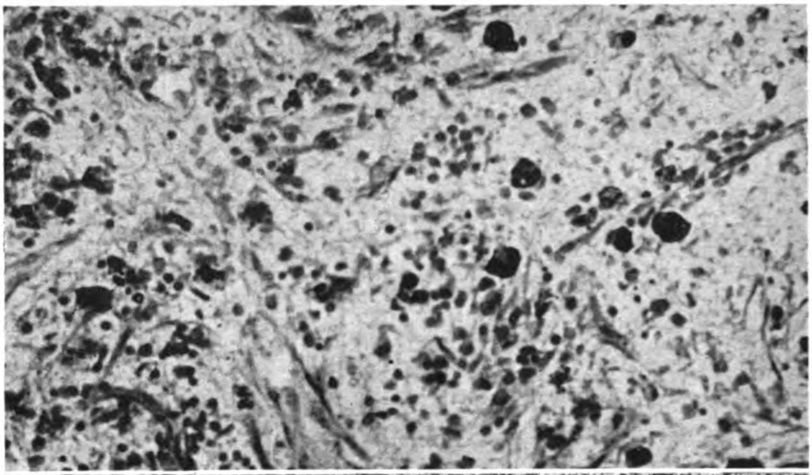


Fig. 13.

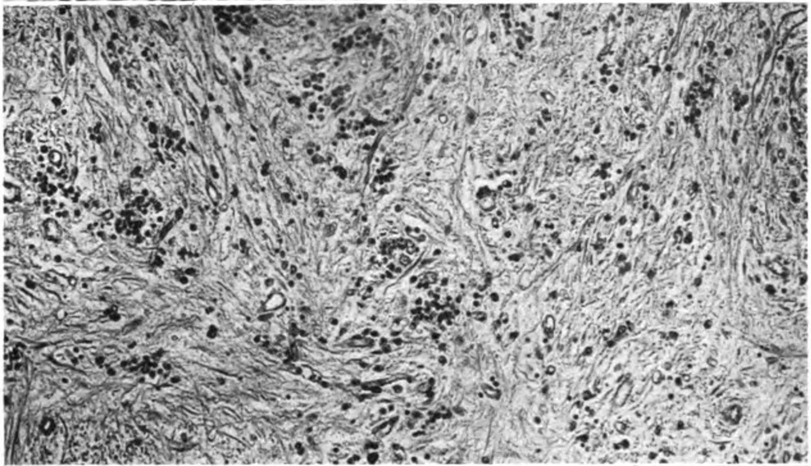


Fig. 14.

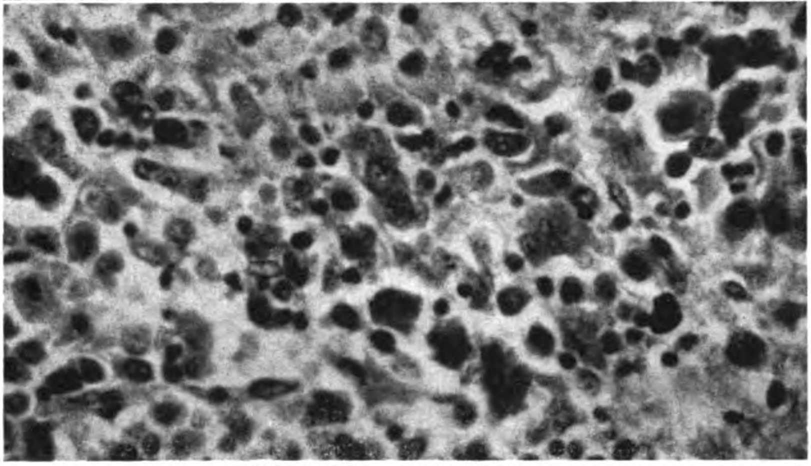


Fig. 15.

*(For legends, see opposite page.)*

Yet 41.8 per cent of them were found to have red corpuscles larger than 94 cubic microns. In only three of the sixteen cases in this plant, with less than 5,000 leucocytes, were the erythrocytes normal in number. This indicated to Greenburg and associates that with benzene absorption the mean corpuscular volume is definitely increased in a large proportion of cases. Regarding the *mean corpuscular hemoglobin,* they estimated the hemoglobin concentration by the constant method of Wintrobe rather than by the usual method calculated in terms of color index. They found that the erythrocytes of benzene workers are likely to contain more than normal amounts of hemoglobin. Calculation of the mean corpuscular hemoglobin concentration shows that, in addition to the greater content of hemoglobin, there is also an increased concentration.

SIGNIFICANT HEMATOLOGIC ABNORMALITIES.—The incidence of significant blood abnormalities found in the series studied by Greenburg and co-workers is revealed in Table VIII.

TABLE VIII. INCIDENCE OF SIGNIFICANT BLOOD ABNORMALITIES IN MEN EXPOSED TO BENZENE

| TEST | CRITERIA OF ABNORMALITY | TOTAL TESTS MADE | ABNORMAL | |
|---|---|---|---|---|
| | | | NUMBER | PERCENTAGE |
| Erythrocytes | Less than 4.5 million | 159 | 76 | 47.8 |
| Mean corpuscular volume | More than 94 cubic microns | 103 | 48 | 46.6 |
| Platelets | Less than 1,000,000 | 107 | 35 | 32.7 |
| Hemoglobin | Less than 13.0 Gm. per 100 cc | 235 | 36 | 15.3 |
| Leucocytes | Less than 5,000 | 332 | 48 | 14.5 |

Erf and Rhoades[6] investigated benzene poisoning in six rotogravure printers, one chemist, one beauty parlor worker, and one rubber cement worker. Concerning the *leucocytes,* they observed that early, when the bone marrow was hyperplastic, the presence of myelocytes and irritated leucocytes was noted, but later, when the marrow became aplastic, there was a decrease in the white cells.

In fifty-two patients tested for *bleeding time* by Erf and Rhoades,[6] they noted an excessively long bleeding time in only two cases and

---

Fig. 13.—Bone marrow (vertebral): Fat is no longer present. There is early infiltration with thin strands of fibrous tissue. The sinusoids are all dilated but some are filled with normoblasts and scattered stem cells and others only with non-nucleated erythrocytes. The large dark-staining elements are hemosiderin-laden phagocytes. (×300.) (Mallory: J. Indust. and Hyg. Toxicol. 21: [Oct.], 1939.)

Fig. 14.—Bone marrow vertebral: The marrow shows dense whorl-like fibrosis with no fat and only a few persistent distorted sinusoids. These are filled with small islands of regenerative hematopoiesis among which normoblasts predominate. (×200.) (Arch. Path. 25: 322, 1938.)

Fig. 15.—Bone marrow (femoral): Fat is completely replaced by cellular marrow. The wide-range of polycellularity and resemblance to Hodgkin's sarcoma are apparent. There is an admixture of stem cells, phagocytes, normoblasts, and multinucleated giant cells. Only a rare granulocyte can be detected. A background of thin fibrous stroma may be observed. (×750.) (Mallory: J. Indust. and Hyg. Toxicol. 21: [Oct.], 1939.)

Fig. 16.

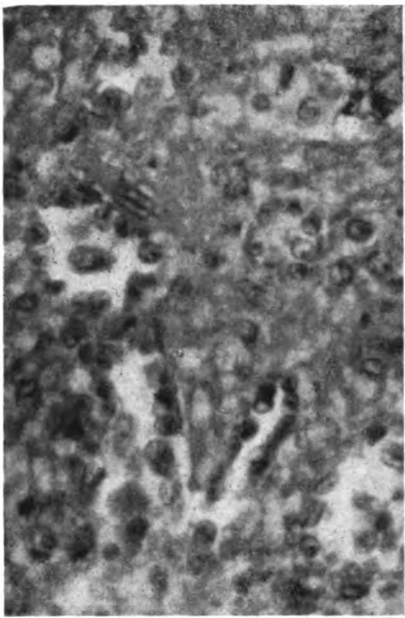


Fig. 17.

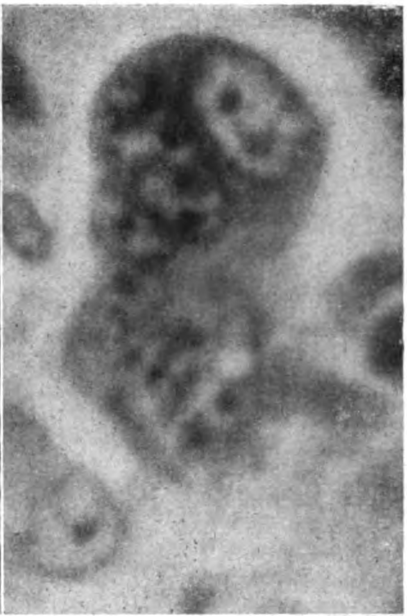


Fig. 18.

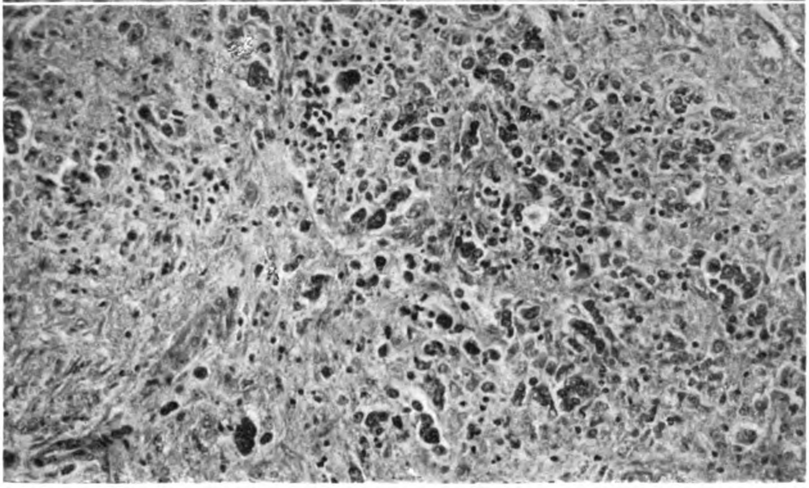


Fig. 16.—Spleen: Pulp lymphocytes have almost completely disappeared. Billroth's cords exhibit marked hemorrhagic extravasation and contain only a few scattered lymphoid elements. Sinusoids are small, but littoral cells protrude prominently into their lumens. (×500.) (Mallory: J. Indust. Hyg. and Toxicol. **21**: [Oct.], 1939.)

Fig. 17.—Spleen: A multinucleated giant cell is shown almost completely filling a splenic sinusoid. Stem cells and normoblasts may be seen in immediate propinquity. (×1000.) (Arch. Path. **25**: 322, 1938.)

Fig. 18.—Spleen: A section demonstrating the resemblance to Hodgkin's disease. There is dense fibrosis and apparent loss of architecture although a few distorted sinusoids filled with stem cells may be seen. Two arterioles are evident as the sole remnants of sclerosed malpighian corpuscles. In the scarred stroma are a variety of cells among which multinucleated giant cells are particularly noteworthy. The small dark-staining round cells which simulate lymphocytes at this magnification are actually normoblasts. (×300.) (Mallory: J. Indust. Hyg. and Toxicol. **21**: [Oct.], 1939.)

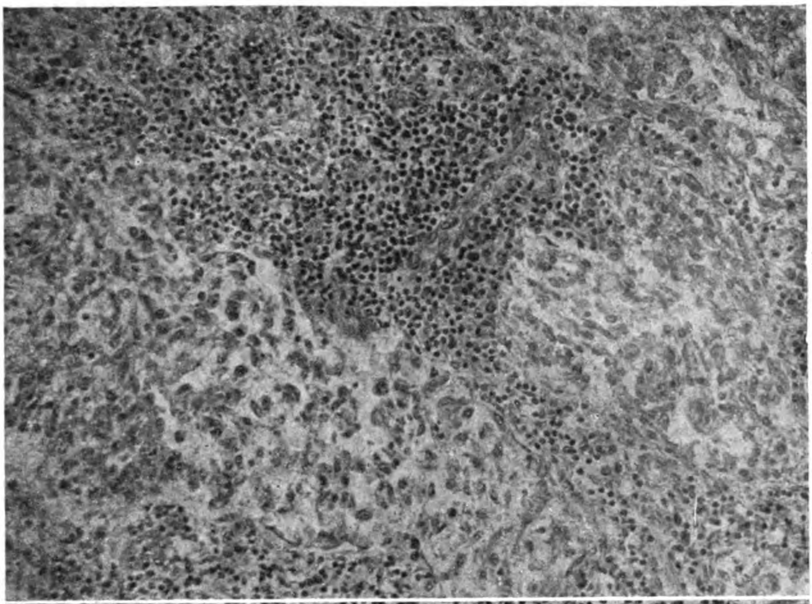


Fig. 19.

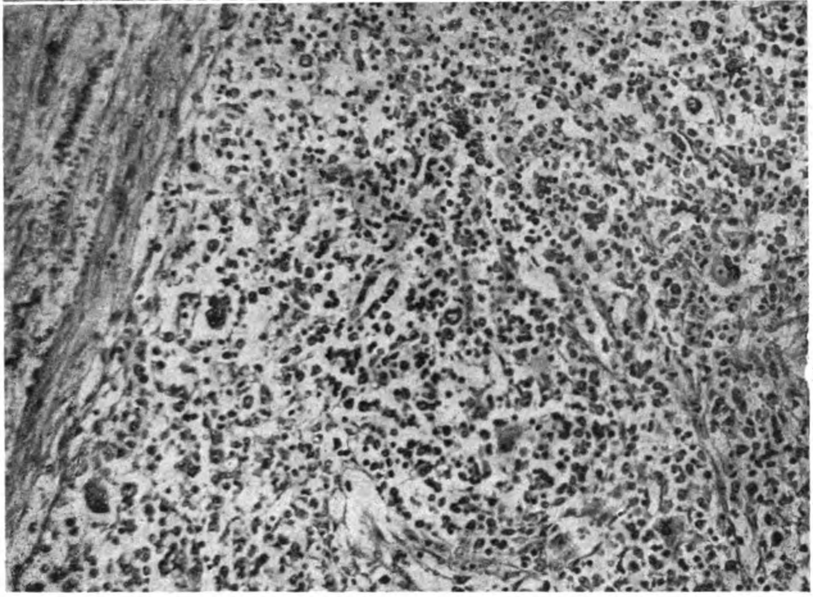


Fig. 20.

Fig. 19.—Lymph node: Lymphoid cords are shrunken and irregular in appearance. The total lymphocytic content is diminished. Sinuses are widely dilated and filled with interlacing strands of spindle-shaped phagocytes. (×250.) (Mallory: J. Indust. Hyg. and Toxicol. **21**: [Oct.], 1939.)

Fig. 20.—Lymph node: A section through the cortex shows apparent obliteration of the subcapsular sinus and invasion of the capsule. Radial sinuses are compressed and distorted as the result of hyperplasia of the pulp. The latter contains, in addition to densely packed lymphocytes, large numbers of all varieties of hematopoietic cells. Multinucleated giant cells may be readily identified. (×350.) (Reproduced through the courtesy of Dr. Ellis Kellert, Ellis Hospital Laboratory, Schenectady, N. Y.)

Fig. 21.

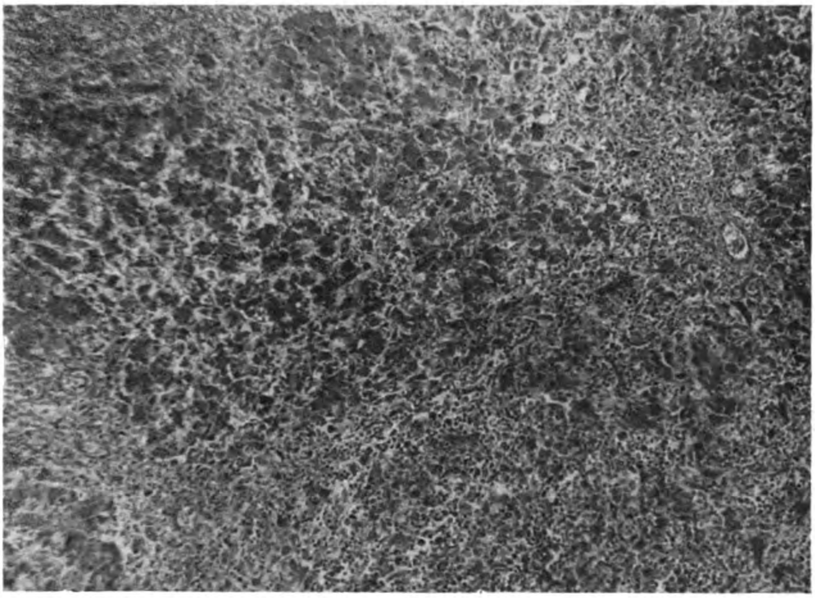


Fig. 22.

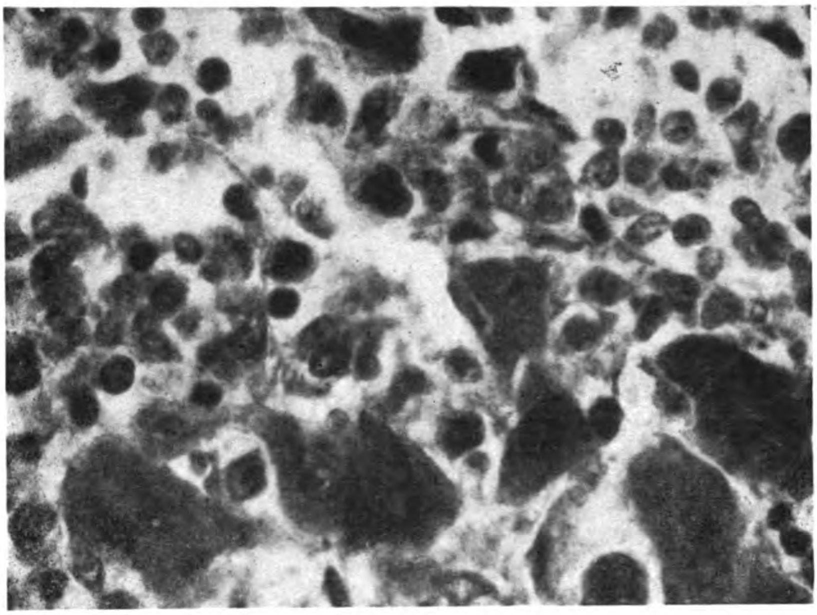
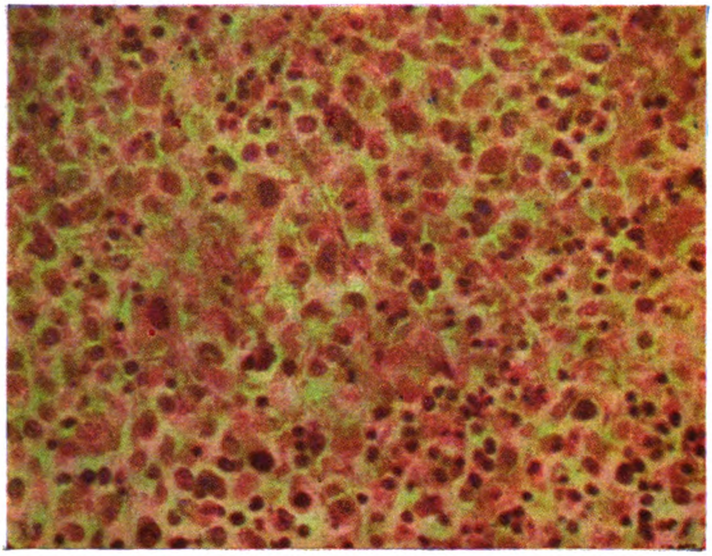


Fig. 21.—Liver: The illustration represents the edge of the 4 cm. tumor nodule noted in the gross. Broad sheets of leukemic cells may be seen invading and destroying liver parenchyma. ( × 150.) (Mallory: J. Indust. Hyg. and Toxicol. **21**: [Oct.], 1939.)

Fig. 22.—Liver: A higher power view of the section shown in Fig. 12. Invasion beyond the confines of the liver sinusoids by leukemic cells may be observed. These cells are almost all myelocytes although identification is not possible in the illustration. Among them, however, small numbers of normoblasts may be identified. Parenchymatous cords show obvious disruption. ( × 600.) (Mallory: J. Indust. Hyg. and Toxicol. **21**: [Oct.], 1939.)

Fig. 23.



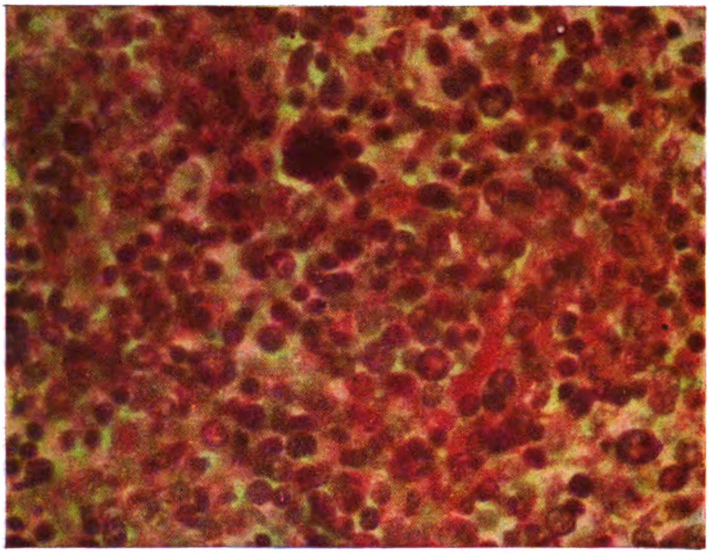
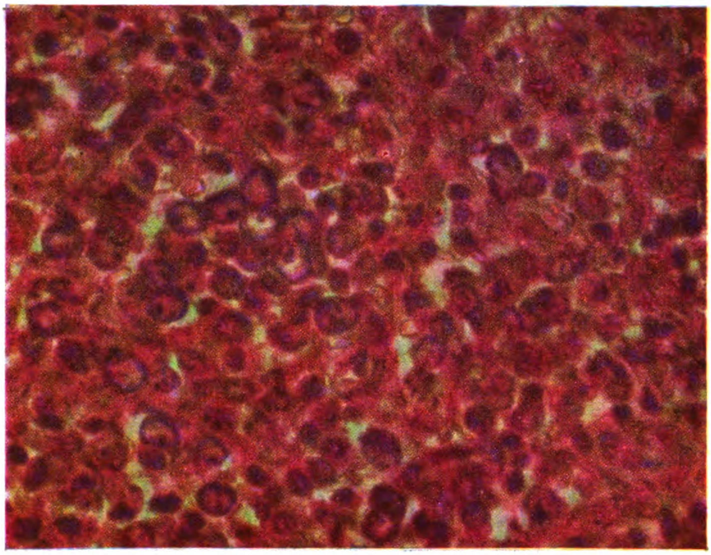


Fig. 24.

Fig. 23.—Bone marrow (femoral): Sinusoidal margins are obscured. There is marked cellularity with erythrogenic elements predominating. Normoblasts and erythroblasts are readily identified. There are also moderate numbers of phagocytes and megakaryocyte-like cells, one of which contains a mitotic figure. (×300.) (Mallory: J. Indust. Hyg. and Toxicol. **21**: [Oct.], 1939.)

Fig. 24.—Spleen: Architecture is partially obscured by a marked accumulation of stem cells and normoblasts. These appear to lie (for the most part) within barely visible distorted sinusoids. A large multipolar mitotic figure is evident. (×600.) (Mallory: J. Indust. Hyg. and Toxicol. **21**: [Oct.], 1939.)

Fig. 25.



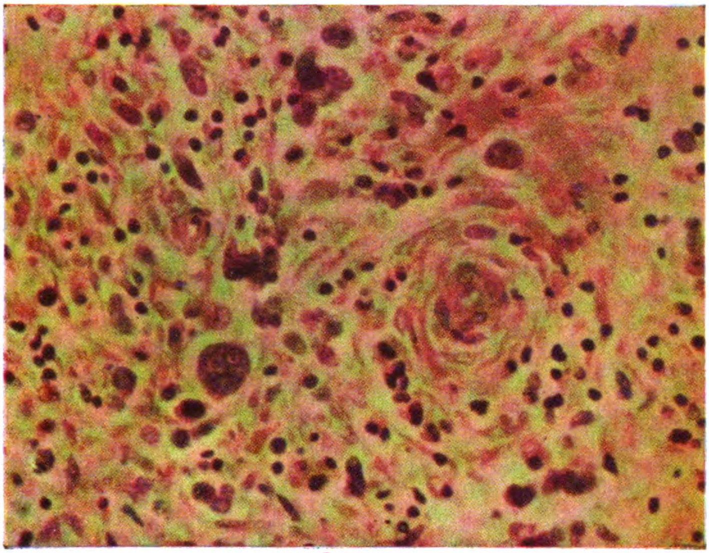
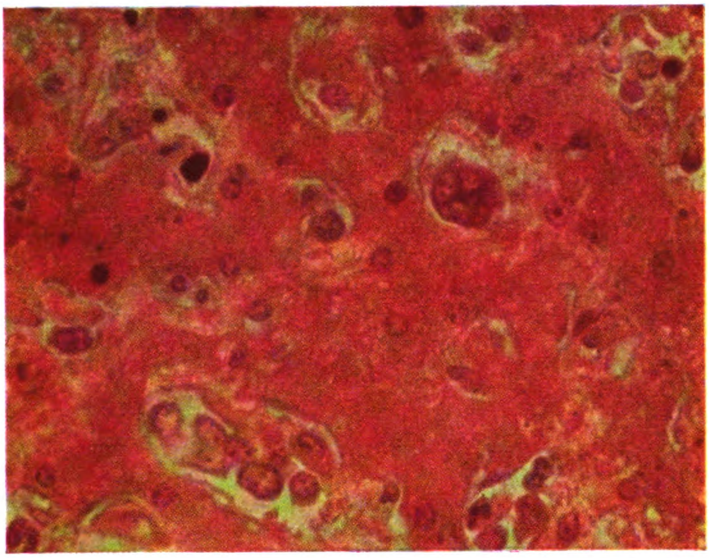


Fig. 26.

**Fig. 25.—Spleen:** Large numbers of stem cells fill the sinusoids and appear also in the intervening pulp. The cells are arranged in irregular clusters. Nuclear vesicularity and relative lack of differentiation are apparent. (×1000.) Mallory: J. Indust. Hyg. and Toxicol. **21**: [Oct.], 1939.)

**Fig. 26.—Spleen:** A sclerotic malpighian corpuscle is demonstrated. Only the central arteriole is recognizable, the lymphoid substance of both corpuscle and parenchyma having been replaced by fibrous tissue. In the region of the arteriole, the collagen is concentrically arranged, and a few polymorphonuclears and residual lymphocytes are seen. Elsewhere there are scattered normoblasts, stem cells, clasmatocytes and multinucleated giant cells. (×400.) (Mallory: J. Indust. Hyg. and Toxicol. **21**: [Oct.], 1939.)

Fig. 27.



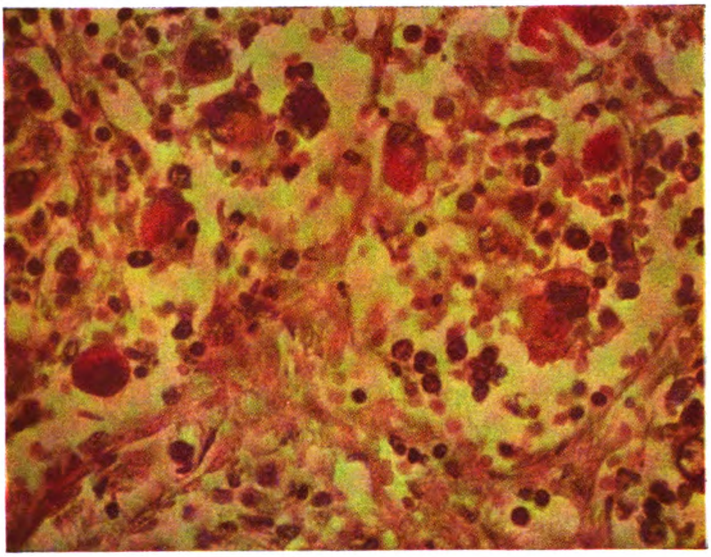


Fig. 28.

**Fig. 27.—Liver: The sinusoids contain clusters of hematopoietic cells. A group** of stem cells and a multinucleated cell indistinguishable from a megakaryocyte are seen. (×750.) (Mallory: J. Indust. Hyg. and Toxicol. **21**: [Oct.], 1939.)

Fig. 28.—Lymph node: A higher power view from the section shown in Fig. 20. Large numbers of multinucleated giant cells may be seen in the midst of a relatively fibrous stroma. Some of these cells are indistinguishable from megakaryocytes. One contains a large mitotic figure. (×500.) (Reproduced through the courtesy of Dr. Ellis Kellert, Ellis Hospital Laboratory, Schenectady, N. Y.)

moderately prolonged bleeding time in only three, although the blood platelets were below 100,000 in thirty-five cases and below 50,000 in twelve others. This group noted no significance in the *sedimentation rate* of men exposed to benzene. Erf and Rhoades[6] made a noteworthy observation when they studied the *free hydrochloric acid content* of the stomachs of their nine patients and found it to be present in every one. This finding should have some value in the differential diagnosis of pernicious anemia from benzol poisoning.

Thrombocytopenic purpura due to benzol poisoning was reported by Vaughan.[8] The occupational history, physical examination, and laboratory studies were as follows:

> "At time of entry the patient was stitcher in a clothing factory. For sixteen years, between the ages of 14 and 31, the patient worked in various shoe factories as a cementer. She handled all types of cements, which dried on her hands during the course of her work. Just prior to her first episode of purpura, the patient had terminated a four months' period of cementing in a factory reported not to be using benzol as a solvent.
>
> "*Physical examination* revealed a well developed, well nourished obese female with a diffuse purpuric rash on the lower extremities. Occasional maculae were also present on the face, back of neck, and dorsum of the hands. Areas of gross ecchymosis appeared in both antecubital spaces following venepuncture. Teeth were carious. The gums were not bleeding on admission. Heart and lungs were negative. The liver edge was felt two centimeters below the costal margin on deep inspiration, and the spleen was barely palpable. There was a definite increase in the area of splenic dullness. There was a slight vaginal discharge. Neurological examination was negative.
>
> "*Laboratory studies* showed a red count of 3.6 million, a white count of 5,250. Hemoglobin was 11.5 grams, hematocrit 40. The differential count showed 63 per cent neutrophils, 31 per cent lymphocytes, 4 per cent monocytes, 4 per cent eosinophils. There was a 2.5 per cent reticulocytosis. Urine was normal. Stools were faintly positive for occult blood. Icterus index was 8. Total protein 6.5. Hinton was doubtful.
>
> "Few platelets were seen in the smear, and by actual count numbered 20,000. Bleeding time was 18 minutes, clotting time 11 minutes, by test tube method. Clot retraction was 47 per cent of normal. Tourniquet test gave 159 purpuric spots per square inch, with 10 spots for the control. Prothrombin time was 55 seconds, with control of 45. Ascorbic acid level was normal. Sternal aspiration revealed an essentially normal marrow picture with no increase in megakaryocytes.
>
> "*Course in hospital.* The patient's vital signs remained normal. The vaginal bleeding ceased after one week. The purpura continued, along with mild bleeding from the gums. The patient was placed on iron and vitamins. On the twelfth day methyl testosterone therapy was begun. At the time of discharge the bleeding time was 9 minutes, clotting time 9 minutes. Clot retraction was 55 per cent of normal. Tourniquet test gave only 40 petechiae per square inch. Platelet count was 30,000, red count 3.9 million, white count 5,950."

In discussing the differential diagnosis, Vaughan comments that:

> "Thrombocytopenic purpura of the idiopathic variety may give a clinical picture quite similar to that described above. The following features, present in

our case, are not commonly seen in idiopathic thrombocytopenic purpura, though they may occur in that disorder: onset in middle age, enlarged spleen, normal bone marrow with no increase in megakaryocytes, quick remittance. Only careful evaluation of the history and clinical data can lead one to the diagnosis of benzol poisoning. The blood studies are most important. Emphasized as signs of benzol poisoning are: (1) either an increase or a decrease in red count and/or the white count; (2) elevated reticulocyte count; (3) megalocytosis; (4) eosinophilia; (5) presence of myelocytes; (6) platelet reduction. Besides the thrombocytopenia our patient exhibited to a slight degree all but one of these characteristics. She had a consistent anemia and leukopenia over a three months' period. On several occasions an eosinophilia was noted. Despite marked blood loss the patient's anemia was megalocytic, with mean corpuscular volumes well over 100. It is impossible to neglect these findings in differential diagnosis. The sixteen years of exposure to benzol is most significant, despite the long latent period."

As stated earlier in the discussion, leucopenia was once believed to constitute an early sign of benzol intoxication but has lately been given no emphasis in this respect over other abnormal blood findings. Browning,[9] feels that these early reports of leucopenia due to benzol poisoning represented cases of heavy exposure which are rarely seen today. She presents studies on two groups of cases. Group A had suffered a heavy exposure, and 67 per cent of these patients showed the classical signs of anemia, leucopenia, and relative lymphocytosis. Group B had undergone chronic exposure to smaller amounts, and among these patients leucopenia was not a prominent finding, while a relative lymphocytosis was found to be present in 62 per cent of cases.

**The "Latent Period" of Benzol Poisoning.**—It is believed by some writers that an anemia due to benzol intoxication may set in months or even years after cessation of exposure. This point is not only of clinical interest but is important as well in matters of medicolegal controversy in compensation claims. Bridge[10] suggests that, in a case which he reported, a latent period of intoxication with a duration of eight years existed; Meyer and Ginsberg[11] believe that benzol was responsible for a case of aplastic anemia in which the patient had withdrawn from exposure ten years prior to the onset of illness; Smith[12] is equally convinced that a latent period of a few years intervened in the case of one of her patients; and in the case of Vaughan,[8] which has been previously cited, the alleged latency of the disease is stated to have been two years. These writers intimate that benzol may initiate pathologic changes in the bone marrow, which may progress at a slow rate for months or even years after exposure.

The opinions expressed in such diagnoses, however, cannot be accepted as based upon unquestionable proof. In Bridge's[10] case, for instance, the patient came under observation following the extraction of a tooth, and it is stated in the report that the case would have been diagnosed as agranulocytic angina if there had not existed a history of benzol exposure. In Smith's[12] case, the patient suffered from diabetes mellitus, and five weeks before admission to the hospital, he, too, had a tooth extracted. Two weeks after the extraction of the tooth he had to cease work because of weakness. An analysis of almost every case reported as exhibiting a similar protracted latent period will reveal ample grounds for the suspicion that some preceding infection had taken place. Smith bases her belief in latent tendencies on the supposition that when exposure occurred, damage was inflicted of a "subclinical degree which was not apparent until subsequent demands, made upon the organism by infection, disease or even the wear and tear of life, caused overt failure of the already damaged hematopoietic system to develop."

The serious implications incurred by unwittingly accepting the theory of a possible latent period following a benzol exposure should be obvious. With experienced workers such as Smith,[12] the decision in any given case fortunately need not be a matter of too much concern. However, the factor of coincidence, or even an unrelated etiology may be lost sight of by the inexperienced physician. Furthermore, after a period of several years, it would be extremely difficult in most instances to determine the degree of exposure or even whether or not a true exposure had actually taken place. It is not uncommon that a worker will state that he was employed in a trade or occupation in which the presence of a certain substance constituted a hazard and yet find that his specific duties did not expose him to this substance. This shows that the possible unreliability of the history will have to be taken into consideration in attempting to associate an anemia presenting itself some years after an alleged exposure with the poisoning said to have been sustained at that earlier date. The need for more scientific observations is decidedly urgent regarding this question.

**Summary.**—A review of the investigations previously mentioned, as well as the work of others not cited in these paragraphs, leads to this conclusion: that chronic benzol exposure brings about a varied clinical and hematologic picture, and that anemia, leucopenia, thrombocytopenia, reticulocytosis, high color index, increased fragility of erythrocytes, urobilinuria, and prolonged coagulation time may or may not be present. The bone marrow may be normal, aplastic, hyperplastic, or leucemic. The variation of the findings is due probably to the degree of exposure, length of exposure, and individual susceptibility.

In spite of the varied picture, two facts remain evident: (1) that anemia invariably results from exposure and (2) that leucopenia is not a common early finding. Furthermore, if leucopenia is present, other abnormalities of the blood picture are also seen. Such observations should discourage the confinement of diagnosis to a white cell count alone.

**Signs and Symptoms.**—Acute intoxication may result from exposure to severe concentrations, wherefrom coma and death may result. Exposure to nonlethal concentrations will produce the picture of a benzol "jag" or drunkenness, such as giddiness, headache, vomiting, disturbed vision, incoherence, etc.

Chronic intoxication causes multiple and varied disturbances which are insidious and invariably not recognized by the victim as being related to his exposure: weakness, increasing nervousness, loss of weight, bleeding from the gums, nose, rectum, or uterus, and altered menses in the female. All these symptoms become intensified as the exposure is prolonged.

**Laboratory Tests.**—There are no specific tests. Men daily exposed to benzol should be under constant surveillance, and tests which include the analysis of the working atmosphere, routine *complete* blood counts, and urine-sulfate ratio determinations should be carried out at regular intervals.

Inhaled benzol vapor is partly converted to phenol in the body, and a portion of the phenol is excreted in the urine as phenol sulfate. Normally, organic sulfate makes up 3 to 15 per cent of the total urinary sulfate; but after inhalation of benzol the percentage of urinary organic sulfate rises while that of inorganic sulfate falls. The sulfate index, which is the percentage of total sulfate present as inorganic sulfate, will decrease. This test is of value only if exposure to benzol exists at the time when the ratio of the different sulfates is estimated. Many writers believe that it is an indicator of abnormal absorption and signalizes damage in advance of any abnormal blood picture. Others consider it to be of only minor importance and judge that its diagnostic value was nil in cases where the clinical picture had been clear cut.

**Possible Disability.**—From an acute, nonfatal exposure, workmen recover without sequelae and the temporary disability is short. In the chronically exposed workman, recovery takes place after treatment for the anemia becomes effective. Permanent disability is rare. If a malignant type of anemia ensues and is unquestionably related to a benzol exposure, the case should be considered as of occupational origin and therefore compensable. However, the pitfalls attendent upon a similar diagnosis, if based on assumption rather than fact, have been mentioned previously.

**Treatment.**—

PROPHYLAXIS.—*Frequent physical and laboratory examinations should be made.* Constant vigilance by blood examinations and *air analysis* should be the established order wherever benzol constitutes a hazard. The routine white cell count commonly resorted to should be supplanted by a *complete blood count,* for reasons emphasized in the preceding paragraphs. Only those in fine physical condition should be placed where a benzol hazard may arise, and frequently physical, as well as laboratory examinations, are indicated.

Men should be *rotated,* and upon perceiving the least variation from the normal, the worker should be removed from his exposure. Likewise, workers should be instructed to report for examination upon noting *bleeding* from the nose or gums or other mucous membranes, or if unaccountable subcutaneous hemorrhages or discolorations are noted. Safety engineers have done an excellent job of preventing accidents by educating the employees regarding the dangers which exist in certain types of employment. It is high time for a similar educational campaign to be conducted in the various occupational diseases. Many serious cases of poisoning from benzol and other noxious agents could be prevented if the employees themselves knew, or were informed, of certain early signs and symptoms. To find a case of far-advanced anemia in one who had for some time noted bleeding and certain symptoms of general malaise denotes not ignorance on his or her part, but a failure of education or instruction. Both general and local exhaust ventilation may be necessary as well as individual protective methods against both inhalation and injury to the skin.

ANEMIA.—

*Blood Transfusions.*—In those workers who have developed anemias of varying degrees together with other symptoms of benzol poisoning, the most valuable form of therapy is blood transfusions repeated frequently if necessary.

*Liver Extract and Iron.*—Since the anemia is frequently of the macrocytic hyperchromic type, liver extract intramuscularly in large dosages (15 to 30 units daily for four days, then every two or three days) or ventriculin orally (15 to 30 Gm. daily) sometimes both should be tried. If the anemia appears to be of a secondary type, the use of iron in the form of ferrous sulfate (5 grains; 0.32 Gm.), two or three times a day, is advised. Leucopenia indicates the trial of pentnucleotides intramuscularly (10 to 40 cc. daily), although marked improvement under this form of therapy has not usually been demonstrated in these cases. It has not yet been determined whether folic acid would be beneficial in toxic anemias.

ROENTGEN RAYS.—For further stimulation of the blood-forming organs, the use of roentgen therapy to the long bones and spleen and bone marrow extracts has been suggested. Others have used ascrobic acid in moderately large doses with reported success.

DIET.—The diet should contain nourishing food with an excess of animal fats and a high calcium content. Calcium preparations may be given orally as supplements to the diet. Intravenous infusions of from 1,000 to 3,000 cc. daily of from 5 to 10 per cent dextrose in saline are of value. Good results from the use of ascorbic acid in daily doses of from 200 to 400 mg. intravenously and orally, until a normal vitamin C level is attained. It is claimed that benzol poisoning is accompanied by a decrease in the content in the urinary excretion of ascorbic acid, and that this disturbance of vitamin C metabolism is not only an accompanying symptom of the toxic process, but also a causative factor in the marked variation of the blood picture.

**Case Histories.**—

"CASE 1.—W. D. S., fifty-four, male, a factory worker, entered the hospital complaining of bleeding from the gums. For seven years the patient had operated a spreading machine in an artificial leather factory, in the process of which he was constantly exposed to benzol fumes. About a year before entry he first noted occasional bleeding from the gums and a slight productive cough.

"*Present Illness.*—For about two months there was slight swelling of the ankles and puffiness of the eyelids, associated with polyuria and polydipsia. For six weeks there was constant bloody oozing from the gums.

"*Physical Examination.*—Examination on admission showed pallor, marked hemorrhagic oozing from the gums, and numerous purpuric spots on the skin. The tip of the spleen was barely palpable, and the liver edge extended 6 cm. beneath the costal margin.

"*Laboratory Examination.*—Urine was normal. Red blood count was 1,900,000, with 45 per cent hemoglobin. The white cells numbered 2,800 with 16 per cent polymorphonuclears, 52 per cent lymphocytes, 8 per cent monocytes, 4 per cent eosinophiles, and 14 per cent basophiles. There was marked stippling of red cells, occasional nucleated red cells, and rare myeloblasts. Platelets were markedly reduced in numbers. Reticulocytes were 2 per cent. Bleeding time was over twenty minutes, and there was no clot retraction in forty-eight hours. The icterus index was 8 units.

"The patient was given five transfusions without improvement. He died on the twenty-eighth hospital day.

"*Necropsy.*—(a) *Skin*—Icteric, multiple petechial hemorrhages. Edema of both lower extremities. (b) *Lungs*—Pulmonary edema and hemorrhagic bronchopneumonia with red blood cells and fibrin but relatively few polymorphonuclears. (c) *Liver*—2,095 Gm. Kupffer cells markedly prominent and filled with hemosiderin. Islets, normal. Within the sinusoids are scattered immature blood cells and moderate numbers of clasmatocytes. (d) *Spleen*—480 Gm. Corpuscles in normal numbers but compressed by surrounding collars consisting of dense aggregations of indeterminate stem cells accompanied by a few normoblasts and phagocytes. Splenic cords are widened and contain hemorrhagic extravasation and hemosiderin-laden phagocytes. Lining cells of sinusoids are prominent. Many of the sinusoids are filled with stem cells, erythroblasts, and occasional normoblasts. There are also some clasmatocytes with both red and white cells ingested. Architecture is generally preserved save for a few foci in which masses of stem cells obscure the structure. (e) *Bone Marrow*—Markedly increased cellularity in both rib and femoral marrow. No fat remains, and there is no evidence of fibrosis. Architecture is obliterated by large clusters of stem cells, erythroblasts, and normoblasts, although moderate numbers of myelocytes and polymorphonuclears could also be observed. Megakaryocytes were rare, mitotic figures abundant, and occasional giant cells with monstrous nuclei could be seen. Phagocytes with hemosiderin were abundant."

"CASE 2.—Male, aged forty-seven. Admitted May 19, 1938, complaining of weakness and ecchymosis. The patient had worked for a rotogravure printing company for three years, and only during the month preceding admission had he noticed increasing weakness and fatigability. He had used solvents containing benzol and was constantly exposed to the fumes. Ten days before admission, subcutaneous ecchymosis appeared on the extremities.

"*Physical Examination.*—The patient was a well-developed and well-nourished male, showing no evidence of recent ecchymosis or purpura. The oral mucous membranes appeared normal, and the liver extended two fingerbreadths below the right costal margin. There were no other pertinent findings.

"*Laboratory Examination.*—Erythrocytes, 2,700,000; hemoglobin, 72 per cent; leukocytes, 2,450; platelets, 50,000; reticulocytes, 6.2 per cent. Differential count: polymorphonuclears, 70 per cent; eosinophiles, 2 per cent; lymphocytes, 20 per cent; monocytes, 8 per cent. Fragility, normal. Icterus index, 8. Fasting gastric contents contained free hydrochloric acid. Urea clearance 135/120 per cent of normal. Urobilinogen excretion: fecal, 410 mg. and 336 mg. excreted daily during two periods; urine,3.8 mg. and 1.1 mg. daily for two periods. A sternal bone-marrow puncture was done, and the marrow was found to be normal. Supravital differential count of the living marrow cells: neutrophilic myelocytes, 22 per cent; myeloblasts, 2 per cent; polymorphonuclears, 21 per cent; normoblasts, 45 per cent; erythroblasts, 10 per cent; Wassermann test, negative.

"*Course.*—Uneventful. The patient received liver extract and thiamine intramuscularly for three weeks in the hospital and three months in the Out-Patient-Department.

"*Laboratory Examination Four Months After Admission.*—Erythrocytes, 3,830,000; hemoglobin, 95 per cent; leucocytes, 7,700; platelets, 256,000; reticulocytes, 4.8 per cent. Differential count: polymorphonuclears, 52 per cent; eosinophiles, 4 per cent; lymphocytes, 38 per cent; and monocytes, 6 per cent. Serum bilirubin, 0.85 mg. per cent. Urobilinogen excretion: fecal, 191 mg. and 192 mg. excreted daily during two periods; urine, 0.8 mg. and 0.6 mg. daily during two periods. The patient recovered fully and was followed over a period of one year with no recurrence or symptoms."

## Toluene

Toluene ($C_6H_5CH_8$) known as toluol and also as methyl benzene, is a colorless liquid with an odor similar to benzol. It boils at 110°C. It is miscible with alcohol, chloroform, ether, acetone and most of the other commonly used industrial solvents. Commercial toluene invariably contains a certain percentage of benzol.

Toluene has more industrial uses than could be listed in the limited space available here. The most frequent of these uses, however, are in the manufacture of dyes and explosives, in the preparation of benzoic acid, or benzaldehyde, as a solvent for the extraction of a great variety of substances, and as a diluent in lacquers, paints, and thinners.

Toluene is a stronger irritant to the mucous membranes than is benzol, but it does not act as a narcotic. Acute poisoning causes a depressant action upon the central nervous system. It is generally believed that chronic exposure to amounts maintained within the permissible limits of concentration does not produce pathologic lesions. Above the allowable limits the effects from exposure to toluene are not conclusive. Greenburg and co-workers[13] studied 106 workers in an airplane plant, who had been exposed to the inhalation of toluene of between 100 and 1,100 p.p.m., for periods of time ranging from two weeks to more than five years. The workers studied were engaged in spray painting, dip painting, and brush painting. To evaluate their findings these investigators used control groups who had not been exposed to toluene.

Sixty-one painters had had no exposure to other solvents; forty-five had had exposure to other solvents, thirty of them to benzene. The comparison with the controls ran as follows: enlarged liver—painters, 30 per cent; controls, 7 per cent. In the sixty-one with no previous exposure, only 21.4 per cent had enlarged liver, but even this is three times the control rate. There was in the painters' group a slight decrease in the red cell count and a slight lymphocytosis; the mean corpuscular volume was somewhat increased in 23.6 per cent (controls 7.2 per cent); the hemoglobin was higher; values of 16 Gm. per 100 or more were found in 37.7 per cent as contrasted to 2.4 per cent of the controls. All these deviations from the normal were slightly more marked in men who had had previous exposure to benzene.

Greenburg and co-workers[13] in reviewing their findings commented as follows:

> "It appears from these data that the results of chronic exposure to toluene of the varying intensities noted produced some objective abnormalities but practically no symptoms. The absence of severe illness was a surprising revelation in view of the fact that the concentration of toluene in the inspired air reached the relatively high average level of 1,100 parts per million and that the period of exposure exceeded five years in many instances. In no instance was it felt necessary to remove an exposed worker from his occupation because of the inhalation of toluene.
>
> "More specifically, there were observed certain abnormalities which, in the light of previous experience, must be interpreted as evidence of mild intoxication. These abnormalities were enlarged liver, mild depression of the erythrocyte level, elevation of mean cell volume, absolute lymphocytosis, elevation of the hemoglobin value, elevation of the mean corpuscular hemoglobin and elevation of the mean corpuscular hemoglobin concentration.
>
> "The findings of hepatomegaly are of special importance, since such a finding as a result of exposure to toluene appears not to have been heretofore described.
>
> "Since no correlation was found between enlargement of the liver and either clinical or laboratory evidence of disease, the possibility suggested itself that the enlargement of the liver might be compensatory in character rather than an indication of hepatic disease. The data collected in the present study, however, *are considered insufficient* to provide an answer to this question.
>
> "The decrease of the erythrocyte levels was of only a moderate degree. This is in strong contrast with observed effects of inhalation of benzene. The increase in absolute lymphocyte counts has frequently been described in exposures to various toxic substances in industry and requires no further comment at this time. The same may be said about the elevation of mean corpuscular volume.—Stress should be placed on the fact that leucopenia was not observed."

TABLE IX. ABNORMALITIES OBSERVED AMONG THE SIXTY-ONE WORKERS IN THE UNCOMPLICATED EXPOSURE GROUP

| CASE | LIVER ENLARGEMENT IN FINGERBREADTHS BELOW COSTAL MARGIN | ERYTHROCYTE COUNT BELOW 4.5 MILLION | MEAN CORPUSCULAR VOLUME ABOVE 99 CUBIC MICRONS | LYMPHOCYTOSIS |
|---|---|---|---|---|
| 2 | 1 | .... | .... | ..... |
| 7 | 0 | 4.26 | 106 | ..... |
| 26 | 0 | .... | .... | 5.525 |
| 27 | 0 | .... | 103 | 5.733 |
| 28 | 0 | 4.35 | 110 | ..... |
| 30 | 0 | 4.32 | 106 | 5.980 |
| 31 | 0 | .... | .... | 5.250 |
| 42 | 0 | .... | .... | 6.392 |
| 51 | 4 | .... | .... | ..... |
| 53 | 4 | .... | .... | ..... |
| 54 | 0 | 4.43 | 106 | ..... |
| 57 | 0 | .... | .... | 5.014 |
| 58 | 1 | .... | 103 | 5.617 |
| 62 | 0 | .... | 108 | ..... |
| 73 | 4 | .... | 100 | ..... |
| 83 | 4 | 4.47 | 103 | ..... |
| 99 | 4 | .... | .... | 5.085 |
| 101 | 2 | .... | .... | ..... |
| 102 | 0 | .... | 102 | ..... |
| 104 | 4 | .... | .... | ..... |
| 107 | 0 | 4.24 | 106 | ..... |
| 113 | 2 | .... | .... | ..... |
| 116 | 0 | 4.38 | .... | ..... |
| 119 | 3 | .... | .... | ..... |
| 121 | 3 | .... | .... | ..... |
| 123 | 4 | .... | 105 | 8.290 |
| 126 | 0 | .... | .... | 5.868 |
| 128 | 0 | .... | .... | 6.420 |
| 129 | 0 | 4.26 | 101 | ..... |

Further studies on toluene were reported by von Oettingen, Neal, and Donahue[14] who tested concentrations of toluene from 50 to 800 p.p.m. on three normal subjects and concluded that inhalation of 200 p.p.m. for eight hours causes slight but definite impairment of coordination and reaction time, which is liable to increase the danger of accidents; with higher concentrations these effects increased, and at 600 to 800 p.p.m. they could be observed after a few hours' exposure. The elimination of hippuric acid in the urine increased with the concentration of toluene in the air, but the ratio of inorganic to organic sulfates was not affected by toluene, nor was there any sign of injury to the blood-forming organs.

The latest study of toluene exposure in a large group is that of Wilson,[15] who was able to follow the history of some thousand employees working with commercial toluene for one to three weeks in concentrations from 50 to 1,500 parts per million. One hundred showed symptoms severe enough to be sent to the hospital for examination, and ten of these showed blood changes. There were no deaths. Those exposed to concentrations no higher than 200 p.p.m. were practically unaffected;

at 200 to 500 p.p.m. headache was complained of, and there developed nausea, anorexia, bad taste in the mouth, lassitude, and slight but definite impairment of coordination and reaction time, but no significant physical or laboratory findings were noted. When the concentration of toluene was over 500 p.p.m., all the disorders mentioned were increased, sometimes markedly, and in several cases petechial hemorrhages appeared under the skin.

In most of the cases the blood picture was normal except for a fall in the red cell count, usually down to 2,500,000. Leucopenia was found in only two cases, with white cell counts of 2,500 to 3,000; here all the other blood elements were reduced, and biopsy of the bone marrow disclosed partial destruction of the blood-forming elements.

### Xylol

Xylol or xylene, $C_6H_4\ (CH_3)_2$, has long been considered less toxic than either benzol or toluene. Usually when mention is made of its injurious effect upon the body, the blame is placed upon the other two members; that is, particularly upon benzol and, to a lesser degree, upon toluene. Rarely has anyone studied the effects of xylol alone. In 1943 Browning[9] reported her observations of a series of cases where the exposure had been to pure xylol, used as a cold solution for an urea formaldehdye polymer. Of these workers, 54.5 per cent showed leucopenia, while 36 per cent suffered from a high degree of relative lymphocytosis.

### Solvent Naphtha

It is well to call attention to the difference between solvent naphtha and naphtha. The word naphtha as ordinarily used refers to the petroleum naphtha of the aliphatic hydrocarbons. In contrast, solvent naphtha is an aromatic hydrocarbon consisting of a mixture of benzol, xylol, toluol, ethyl-benzene, and some higher hydrocarbons. It is used as a diluent in lacquers, paints, and varnishes and as a solvent for gums, resins, etc. Its toxic action and physiologic response are due to its components; that is, benzol, toluol, and xylol.

### Trinitrotoluene

Trinitrotoluene, $C_6H_2CH_3(NO_2)_3$, familiarly known as TNT, is a pale yellow, crystalline substance having high explosive properties. It is soluble in oils and greases as well as in ether, acetone, alcohol, and various solvents. It melts at 80°C., at which time it gives off a pungent odor similar to that of bitter almonds.

During World War I 17,000 cases of TNT poisoning occurred in the United States, with 475 fatalities. In World War II the total number of fatalities reported from all government-owned ordnance plants was twenty-two, which, according to McConnell and Flinn,[16] is a rate of less than 3 per 100,000 operating employees per year. This gratifyingly low rate of mortality testifies to the excellent industrial hygiene established by McConnell and associates.

In addition to affecting the skin, there are two other disturbances arising out of TNT poisoning; namely, toxic hepatitis and aplastic anemia. Trinitrotoluene may be inhaled, ingested, or absorbed through the skin. The clinical picture is that of fatigue, headache, anorexia, lassitude, nausea, abdominal pain, and tightness in the chest. Cyanosis is a characteristic sign, as evidenced by the bluish discoloration of the lips and ear lobes. With the involvement of the liver, jaundice will appear. As a rule, victims of TNT poisoning suffer either from toxic hepatitis or aplastic anemia, but the combination of these two conditions in one individual is rare.

According to McConnell and Flinn,[16] factors other than the concentration of trinitrotoluene are responsible for this type of poisoning.

Only a third of the total number who died were exposed to average concentrations in excess of the generally accepted maximum allowable concentration of 1.5 mg. of TNT per cubic meter of air, and a few of these probably wore respirators, so that the average amount of TNT in the respired air was not relatively high. A substantial number of employees not clinically affected were similarly exposed. However, it cannot be stated how much of the material was ingested or absorbed through the skin, the quantity depending largely on the personal habits and hygiene of the individual. It appears quite probable that aside from the total amount of TNT entering the body, individual differences, undernourishment or nutritional disturbances, excesses, acute illnesses, and other factors may render the individual more susceptible to the toxic action of TNT.

The fact that in this series hepatitis occurred more frequently among the younger age group (average age, 30 years) and aplastic anemia among the older age group (average age, 45 years) suggests that age may be an influencing factor. The proportion of TNT-exposed workers in the various age groups is not known.

The former group also had a much shorter period of exposure than the latter group. Only four of the eight persons dying from hepatitis were exposed to TNT two full months or more, and only two were exposed three full months or more. On the other hand, only one of the fourteen persons dying from aplastic anemia did not survive at least three months' exposure. The median period of exposure for those with hepatitis was 63 days, as compared with 216 days for those with aplastic anemia.

Unfortunately, the early symptoms of poisoning are often indefinite and frequently are not sufficiently marked by the patient to note and report. Although most of the patients in the early stages of toxic hepatitis reported to the plant dispensaries complaining of symptoms of nausea, vomiting, drowsiness, and general malaise, others, oblivious to any subjective feeling of illness, continued to work until jaundice appeared or laboratory tests indicated evidence of toxicity. In those presenting evidence of aplastic anemia, the early symptoms reported consisted of weakness, loss of appetite and weight, mild cough, and bleeding usually from the nose and gums. In several instances symptoms were absent or the patient noticed only a few purple spots on his body which aroused his curiosity.

Although several procedures have proved helpful in diagnosing TNT poisoning, no single test has been found sufficiently reliable for either the early detection of liver damage or aplastic anemia. Perhaps the most consistent laboratory findings, and those generally accepted as disclosing significant evidence, are an elevation of the icterus index for indicating early liver damage, and either a decrease in hemoglobin or total cell pack for anemia. These tests should be made part of the routine monthly medical examination of all TNT-exposed workers unless the plant experience has been favorable, in which case the interval may be extended to six or eight weeks. Other early findings may be significant reductions in the white cell and platelet counts.

An initial high icterus index which rose rapidly was a constant finding in the fatal cases of toxic hepatitis. In only a few instances there was a reduction of the hemoglobin content and the erythrocyte count, and without a marked change of the blood picture. Cyanosis was infrequently noticed. Deep jaundice, with severe weakness, stupor, and other manifestations of acute toxemia developed rapidly and terminated fatally in a relatively short time. In these eight cases the elapsed time from the first definite signs or symptoms to death ranged from twelve to fifty-three days, with a median value of thirty-four days.

In the anemia cases, a rapid reduction of the number of red blood cells and of the hemoglobin are perhaps the most conspicuous signs of significant bone marrow damage. It is not unusual to find a depression of all cellular elements, and a marked reduction in the platelets is a grave sign. A hypoplastic or aplastic appearance of the bone marrow was a common finding. The marked tendency to, or presence of, petechiae is often associated with aplastic anemia. The course of the disease commonly progresses more or less rapidly to fatal termination. In these cases, the elapsed time from the onset of definite symptoms until death ranged from six to 185 days, with a median figure of forty days.

**Laboratory Tests.**—Although not a laboratory procedure, the application of alcoholic sodium hydroxide (Webster) will reveal the presence of even small amounts of TNT on the skin by producing a purplish discoloration. The reagent is made by adding one part by volume of saturated stock solution of sodium hydroxide in absolute alcohol to nine parts by volume of absolute alcohol. Norwood[17] suggests that this test has a valuable psychologic effect on the workers, for it tends to convince them of the need for more rigid personal hygiene.

Horecker[18] feels that the formation of Heinz bodies serves to indicate the level of exposure and possibly the extent of intoxication. Pinto and Wilson[19] described a method for the determination of TNT derivatives in the urine of workers exposed to TNT either by inhalation or through skin absorption. The TNT derivatives are extracted from urine with ethyl ether, reduced to triaminotoluene by titanium trichloride, and after diazotization are coupled with dimethyl-alpha-naphthylamine. Workers exposed to different amounts of TNT during the course of their work have shown variations in the amount of TNT derivatives present in their urine.

Snyder and von Oettingen[20] devised a test based on the extraction of metabolites of TNT with ether, and determination of the amine in the ether extract after evaporation of the ether by diazotization and coupling with alpha-naphthylamine, extraction of the latter with toluene, and comparison of the orange-yellow color formed with a set of standards. In addition, clinical tests, as for instance the hepatic function test, blood studies, bone marrow studies, or the determination of urinary coproporphyrin excretion are valuable.

**Treatment.**—Removal of any TNT particles in the atmosphere by means of proper ventilation is the first major step in safeguarding workers engaged in the manufacturing or other handling of TNT. The workers should be carefully selected and those under weight, those under 20 years of age, those with past medical history of anemia or liver disturbance, those who perspire too freely, nail biters, and those who appear to be habitually indifferent about their personal cleanliness should not be allowed to work with TNT. Protective clothing should be designed to cover as much of the body as possible. Norwood[17] devised an agent to remove TNT from the skin, by combining a wetting agent and potassium sulfite in an oil solution. Workers should be checked at frequent intervals by blood studies and the icteric index, withdrawing them from exposure when alterations from the normal are evident.

Treatment of the poisoned worker includes a high protein and carbohydrate diet, administration of liver extract, blood transfusions, and possible infusions of glucose. Although at the time of this writing the use of folic acid in aplastic anemia has undergone only a limited period of experimentation, it should be tried in cases of TNT poisoning. Pyridoxine in large doses intravenously as well as orally has also been suggested. It is questionable whether methionine would have any value in the treatment of acute hepatitis due to TNT poisoning.

**Case History.**—Up to the present time the only case in the literature of a TNT intoxication which combined evidence of both a toxic hepatitis and aplastic anemia is the following as reported by Sievers and co-workers.[21]

"A 43 year old white farmer was employed at a TNT bomb and shell loading plant on July 27, 1943. The preemployment physical and laboratory examinations disclosed no significant abnormalities. His first period of employment, August to December, 1943, was uneventful except for a tetryl rash which disappeared a few weeks after he was removed from contact with tetryl. He was rehired on April 5, 1944, and put on various jobs involving exposure to TNT such as pushout risers (four days), cleaning rubber funnels (twenty-six days), and steam-out operations (forty-six days); these operations involved an average atmospheric exposure of 4.2, 3.1, and 1.1 mg. of TNT per cubic meter of air, respectively. Another eighteen days were spent on odd jobs free of contact with TNT. On July 24 he reported to the medical station, stating that he could not work in TNT (no reason given). The observations at the time were: blood pressure, 120 mm. of mercury systolic and 76 mm. diastolic; hemoglobin, 15 Gm. per hundred cubic centimeters, and red blood cells, 4,450,000 per cubic millimeter. The only previous incident worth noting was a second degree burn on the right ankle treated with 5 per cent sulfathiazole ointment for nine weeks.

"On August 1 he returned, complaining of nausea, loss of appetite, constipation, sore throat, lumbar pain, burning of eyes and dysuria. His skin and scleras were severely jaundiced, and the liver was enlarged upward. The erythrocyte count was 4,010,000 per cubic millimeter, and the icterus index was 100 units; the urinalysis revealed a dark amber clear acrid urine with a specific gravity of 1.017, a 3 plus reaction for sugar, a 1 plus reaction for albumin and a negative reaction to the Webster test. The patient was hospitalized and placed on a diet high in carbohydrates, high in proteins and low in fat. Additional therapy consisted of frequent injections of concentrated extract of liver (1 cc. of Lederle's extract which contains 15 units per cubic centimeter), a high potency polyvitamin capsule daily, vitamin K, and later ferrous sulfate. The diagnosis at admission was toxic hepatitis with a grave prognosis.

"During the first three weeks of hospitalization, there was little change in the patient's condition. He complained of constant abdominal distress and occasional nausea. The stools were frequently tarry and on one occasion he had an emesis of 'coffee-ground' vomitus. The patient improved from the fourth to the tenth week with respect to hepatic function and gastrointestinal distress. At the end of six weeks the jaundice had practically disappeared, the icterus index had fallen to thirty units, and the patient was comparatively free of complaints.

"However, the red cell count and hemoglobin value had dropped noticeably during the third week in the hospital, without any change in the white cell picture. Two transfusions of fresh whole blood were given four days part, and a third was given seven days after the second transfusion. It became more apparent that the anemia was of the aplastic type on the thirty-third hospital day, when multiple petechiae appeared over the abdomen and chest. A platelet count at this time was 60,000 per cubic millimeter (direct method) but the total and differential white counts remained within normal limits for another month. Bleeding of the gums was first noted on the forty-sixth hospital day and continued with gradually increasing severity until the patient's death.

"On September 29 (the sixty-first hospital day) the patient had severe chills and fever twenty-four hours after his eighth transfusion of whole blood. During the next fourteen days the patient's temperature oscillated between 100 F. and 104 F., his respiratory rate doubled, and his pulse rate was 120 per minute. He complained of general malaise and aching, particularly in the chest and upper part of the abdomen. During the last thirty-six hours of life, the patient became irrational and slightly jaundiced, and several purpural patches appeared over the abdomen and extremities. The pulse rate averaged from 130 to 140 per minute. The blood pressure fell to 86 mm. of mercury systolic and from 45 to 30 mm. diastolic, and crepitant rales were heard at the base of the lungs.

"The therapeutic regimen which has been outlined continued unchanged until October 12, two days before death. At this time additional treatment included 2 mg. of folic acid orally twice daily and intramuscular injections of 10 cc. of pentnucleotide every six hours. A total of 6 mg. of folic acid and 20 cc. of pentnucleotide was administered before death. Five transfusions of whole blood (500 cc. each) were given during the last two weeks with the hemoglobin remaining between 6 and 8 Gm. per hundred cubic centimeters and erythrocyte counts varying from 2,000,000 to 2,800,000 cells per cubic millimeter. The total white cell count dropped to 2,400 cells per cubic millimeter, of which approximately 1,900 were lymphocytes. The platelets were very scarce, and those present were larger than normal. In the sternal blood smear the variation from normal was a pronounced increase in lymphocytes, and a decrease in granulocytes with an increase in young myeloid cells. A few polychromatophilic cells were seen which were often slightly smaller than mature erythrocytes. Stippled erythrocytes were rarely seen, whereas normoblasts and megaloblasts were present in moderate numbers. The few platelets that were present were larger and more granular than usual. Other laboratory observations for the day preceding death were: icterus index 50 units, prolonged bleeding time, normal clotting time but poor clot retraction. The urinary coproporphyrin excretion on October 12 was 958 micrograms per twenty-four hours, and it was 325 micrograms for the twelve hours preceding death on October 13.

"*Observations at Autopsy.*—The autopsy was begun five hours after death. Of the gross examination only the pertinent observations are recorded here. The scleras and skin were moderately jaundiced. There were 20 to 30 purplish, purpuric spots from 3 to 5 mm. in diameter over the abdomen, and a few were scattered over the extremities. The lungs were only slightly collapsed and firmly adherent to the parietal pleura. The left lung weighed 735 Gm. and the right 798 Gm. The lungs were crepitant throughout, and on section a clear frothy fluid oozed from the cut surface. The peritoneal cavity contained 250 cc. of bright yellowish orange fluid. Many petechiae were seen through the serosa of the small intestine and transverse colon. The stomach contained about 200 cc. of dark brown fluid. There were numerous hemorrhagic areas scattered throughout the mucosa of the stomach, intestine and urinary bladder. The liver was enlarged, weighing 2,140 Gm. The surface was moderately pale, yellowish pink, with many scattered raised firm nodules of a lighter color. Both surfaces of the anterior border of the right lobe were demarcated from the rest of the lobe as a firm dark red constricted area about 7 cm. long and 2 cm. wide. The cut surface was nodular and of variable consistency, being mostly friable and soft. There were small dark red depressed areas scattered throughout all sections of the lobes. The lobular markings were indistinct. The spleen was dark purple and friable and weighed 175 Gm. The kidneys were soft, friable and pale. The capsule stripped with ease. A cross section of the sternal bone marrow was pale pink.

"*Microscopic Examination.*—

"*Heart.* Sections showed a few small focal epicardial hemorrhages and slight coronary arteriosclerosis.

"*Lungs.* There were occasional small foci of alveolar exudate and gram-positive cocci.

"*Gastrointestinal Tract.* Foci of old and recent hemorrhage were present in the mucosa of the stomach and intestine.

"*Liver.* Sections showed large areas of collapsed, congested stroma without hepatic cells and without appreciable fibrosis. In addition, the stroma was infiltrated with numerous lymphocytes, plasma cells and macrophages. There was slight proliferation of the bile ducts in portal areas. In such collapsed islets and lobules of hepatic cells, often irregularly arranged, the hepatic cells contained more or less fat globules and a little iron-positive pigment.

"*Spleen.* The red pulp showed irregular congestion and focal infiltration of plasma cells.

"*Bone Marrow.* There were numerous plasma cells of variable sizes, some multinucleated. Normoblasts, granulocytic leukocytes and megakaryocytes were rare. Granulocytes when seen were usually myelocytes and myeloblasts.

"*Kidneys.* There was slight arteriosclerosis with small focal scars and infiltration of lymphocytes and plasma cells. Extensive hemorrhage was noted in the pelvic fat.

"*Bladder.* Focal hemorrhages were present in the various parts of the wall of the bladder.

"The other organs examined, i.e., pancreas, lymph node, adrenal gland and sciatic nerve, presented no significant pathologic condition.

"The diagnosis was (1) subacute ('red') atrophy of the liver and (2) hypoplasia of the bone marrow (aplastic anemia)."

## Tetryl

Tetryl is trinitrophenylmethylnitramine, an important military propellant. Tetryl dermatosis was recognized during World War I as a very troublesome affection, but it was believed that this was the only form of poisoning to be looked for in tetryl workers. More careful studies made during 1943 revealed the fact that systemic effects also may occur. Witkowski and colleagues[22] report no less than 1,258 cases of industrial illness due to tetryl in a force of over 5,000 workers. The chief complaint was of a dermatitis, usually on the face and neck. Epistaxis was a common complaint, and small ulcerations of the nasal mucosa furnished the cause. A systemic effect was also seen. Anorexia, mild nausea, flatulence, and abdominal cramps occurred in 10 per cent of all exposed; dry cough and pain in the chest, headache, irritability, sleeplessness, and lassitude were frequent complaints. Three cases were serious enough for hospitalization. All the patients had a history of severe tetryl dermatitis, which occurred with increased intensity on a second accidental exposure. The authors emphasize also the occurrence of rapidly developing secondary anemia.

## Nitrobenzene

Nitrobenzene, $C_6H_5NO_2$, is used in the manufacture of explosives, but also in perfumes, lacquers, and shoe polish and in combination with certain aniline products. It is absorbed readily through the skin, in which case it usually exercises a delayed action. By inhalation of high concentrations it acts as a depressant on the central nervous system, as illustrated by one case which was brought to the attention of the author.

A chemist's assistant spilled nitrobenzene on his trousers and almost at once fell to the floor. He was rushed to the hospital in his soaking clothes and was unconscious when he arrived with respiration at first rapid, then slow and irregular, pulse feeble, heart sounds distant and weak, and cyanosis marked. A sample of blood was of a deep brown color. He died an hour and a half after the accident. Post-mortem examination revealed nothing of note.

Chronic exposure leads to a blood picture similar to benzol intoxication. Involvement of the central nervous system is manifested by staggering gait, incoherent speech, diminution of vision, somnolence, and vertigo.

### Aniline

Aniline is one of the aromatic amines which is used extensively in the manufacture of dyes and in the rubber industry. The substances used in the production of coal tar dyes, to mention only the toxic ones, are the "crudes," benzene, toluene, xylene, phenol, which are treated with mineral acids, alkalies and certain compounds of the aliphatic series such as methyl alcohol, dimethyl sulfate, formaldehyde, methyl chlorides; reduction is effected by means of nascent hydrogen produced from zinc and hydrochloric acid in the course of which $AsH_3$ may be given off, nitration by nitric acid and oxidation by potassium chlorate and lead peroxide. The resulting compounds, known as "intermediates," are chiefly nitro, chlor, and amino compounds. Reduction is now being replaced by amidation in some plants. Aniline can be produced by amidation of benzene with ammonia, and this process does not carry any danger from $AsH_3$.

A new use for aniline is as a stabilizer of trichlorethylene in degreasing tanks to prevent the break up of tri with the formation of metal-corroding hydrochloric acid.

Aniline affects the nervous system and the blood, causing fatigue, loss of appetite, headache, vertigo, disturbed speech, and varied urinary manifestations. The earlier literature maintains that it has been the cause of bladder tumors. Recent investigations, however, would tend to disagree with the contention that aniline acts as an agent provocative of bladder tumors. At least this much is certain: that the present methods of handling aniline in industry have prevented the occurrence of any recognizable incident.

### Phenol

Phenol, $C_6H_5OH$, known commonly as carbolic acid, is used extensively in the chemical industry and of late has been employed to form one of the basic constituents in the manufacture of plastics. In industry its chief effects is exercised on the skin. Inhalation of its vapors disturbs the central nervous system. Severe absorption may cause death as proved by a case from the author's files:

A 22-year-old male, employed by a concern manufacturing ink, was crossing the plant when he dropped a bottle of carbolic acid. This saturated the right leg, right side of the abdomen, and the chest. Fellow employees immediately removed the shirt and threw water over the upper body. The patient then walked across the street to a physician's office without any difficulty. However, within fifteen minutes after arriving at the doctor's office he collapsed and died.

Autopsy examination, at this clinic, revealed extensive first- and second-degree burns of the right side of the body, right arm, right leg, and scrotum. There was hyperemia and edema of the lower portions of the lower lobes of both lungs, and marked hyperemia and edema of both kidneys, pancreas, and spleen. There was no change in the heart or liver. Analysis of portions of the pancreas, spleen, and kidneys was positive for carbolic acid, and the chemist reported the quantity found was increased five times over that usually found in these tissues at autopsy.

As in any instance where a patient is saturated with a known noxious solution, all clothing should be removed in order to prevent further absorption. The foregoing case illustrates the rapid absorption through the skin and by inhalation, producing death.

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# CHAPTER XVIII

# THE OXIDES OF NITROGEN

Nitrogen forms at least six different oxides, but only three of these are of practical importance. Nitrous oxide ($N_2O$) is a colorless, fairly stable gas, prepared by heating ammonium nitrate. It is used in anesthesia, in admixture with oxygen.

Nitric oxide (NO) is a colorless, slightly soluble gas prepared by the action of copper upon dilute nitric acid. It can also be formed by direct union of nitrogen and oxygen, under the influence of electric sparks, or in an electric arc. The product must be quickly cooled to prevent its decomposition into nitrogen and oxygen. Nitric oxide has some toxic properties, but it is not an irritant. Its inherent danger lies in its possible oxidation to the oxide.

Nitrogen dioxide ($N_2O$) is a gas remarkable for its change of color with the change of temperature. When cooled it is a pale yellow and at room temperature it is a reddish brown, but when heated it becomes deep, chocolate brown. Nitrogen dioxide combines with itself, two molecules uniting to form a molecule of nitrogen tetroxide ($N_2O_4$).

| | | | |
|---|---|---|---|
| | Nitrogen dioxide | | Nitrogen tetroxide |
| *as* | 2 $NO_2$ | Low temperatures<br>$\longrightarrow$<br>$\longleftarrow$<br>High temperatures | $N_2O_4$ |
| | Dark brown | | Nearly colorless |

In whatever molecular form the dioxide is inhaled, it is at once altered into that form corresponding to body temperature. At 40° C. approximately 30 per cent of the dioxide is in the form of $NO_2$ and 70 per cent, $N_2O_4$. It is in this proportion that the gas acts upon the respiratory tract, where it decomposes into nitric acid and nitrous oxide. In reacting with the alkalies in the respiratory tract, a conversion into nitrates and nitrites is brought about. It is the nitrite which, when absorbed, causes a marked systemic reaction.

**Occupational Exposure.**—The possibilities of the presence of undue amounts of nitrogen dioxide are many, if precautionary measures are inadequate. An exposure exists in the manufacture of explosives or in the making of nitrocellulose, in photoengraving and in making photographic films, in metal etching, in pickling, in welding and oxyacetylene operations, as well as in carbon arc booths.

**Pathology.**—If the dosage is overwhelming and death occurs immediately, there will probably be no pathologic changes noted at post-mortem other than congestion of the lungs. In those cases where death was delayed or in the severely intoxicated there is a change of the arterial blood to venous, the presence of hemorrhages into the various organs, edema of the brain, tracheobronchitis, bronchopneumonia, and pulmonary edema. Pulmonary edema is the most frequent finding.

On microscopic study of the lungs, edema of the alveolar wall will be found as well as exudate into the alveolar sacs, swelling, and desquamation of the alveolar epithelium, and polymorphonuclear infiltration.

**Signs and Symptoms.**—It should be emphasized that the *symptoms are usually delayed.* This latent period may be from a few hours to twenty-four or thirty hours. Unquestionably this offers difficulty in some instances in diagnosis, and unless the facts are known the etiology may be overlooked.

ACUTE EXPOSURE.—In the acute nonfatal cases, headache, dizziness, cough, palpitation of the heart and sometimes cyanosis, restlessness, and inability to sleep are common. In certain concentrations, the workman may feel dopey. This narcotic or depressive action sometimes retards the cough reflex, and the patient is not warned of the danger. Pneumonia is a common sequela.

CHRONIC EXPOSURE.—This leads to headache, sleeplessness, loss of appetite and weight, dyspepsia, constipation, and ulcers of the mucous membranes.

DIAGNOSTIC AIDS.—Since pulmonary edema may occur without causing any pain or sufficient discomfort to warn the victim of his predicament, and since therefore a period of time, possibly twelve to twenty-four hours, may elapse in which a suspected case should be kept under vigilant observation, what diagnostic and prognostic procedures may be utilized? According to Fleming,[1] there are two periods of anoxia following nitrous fume gassing. The first follows immediately after exposure and is due apparently to a spasm of the terminal bronchioles and an upset in the Hering-Breuer reflex, interfering with the aeration of the lung and causing acute circulatory collapse. The second period is associated with the development of severe pulmonary edema.

Blood platelet count made on venous blood, if possible, is a very reliable index of the severity of gassing by lung irritants such as nitrous fumes. If a marked elevation in the count takes place immediately after exposure, it is a good indication that severe pulmonary edema will follow.

The changes noted in respiratory rate, blood pressure, the oxygen content of the arterial and venous blood, and the early lung pathology in nitrous fume poisoning are all reliable indices of the degree of poisoning.

In a study at the Haskell Laboratory of Industrial Toxicology, on both dogs and human beings, Fleming[1] noted that an initial rise in the respiratory rate occurred with higher concentrations; and the higher the initial increase, the longer it remained elevated, unaccompanied by increased pulmonary ventilation: the carbon dioxide content of the arterial blood and alveolar air is decreased; an increase in the venous blood platelet count and a fairly marked drop of the systolic pressure together with a moderate drop in the diastolic pressure occurs.

According to conclusions arrived at by the aforementioned author, it is suggested that in a given case where a possibility of exposure exists, but where no immediate signs or symptoms are present, the following observations should dictate further action and influence the diagnosis: if no history of coughing, respiratory distress, nausea, vomiting, headache, or lassitude can be established and no significant changes in the blood pressure, volume index, respiratory rate, and blood platelet count are to be found if observations are made every hour for eight hours after exposure, there seems little likelihood of pulmonary edema, and the patient may be discharged.

If the gassing has been severe, asthmatic breathing may be detected within the first half hour. The *respiratory rate* will be two to three times the normal rate. During the first six hours after exposure, it may drop somewhat, though still remaining far above the normal level, only to rise again if pulmonary edema becomes marked. The *blood pressure* is usually low in the first two to three hours but may become elevated if asphyxia develops.

A *platelet count* made on venous blood is highly significant. Even with exposure to low concentrations of nitrous fumes, the count may be 10 to 15 per cent above normal. If it rises 50 to 100 per cent above normal and continues to rise in the first five hours after exposure, the gassing has been moderately severe.

The exact cause of this marked rise in platelet count is not known. Damage to the alveolar capillaries, with subsequent deposit of large numbers of platelets, many of which may be carried into the blood stream, may be responsible or the associated asphyxia may, in part, be responsible. Tocantins,[2] in a review on blood platelets, mentions a two- or threefold increase in platelets following asphyxia with carbon monoxide or carbon dioxide. Whatever the cause of this marked rise, it is a very valuable index of the severity of gassing by nitrous fumes.

**Treatment.**—Any workman suspected of having been exposed to nitrogen dioxide should be put to bed for twenty-four hours at complete rest and with heat to the body. Estimation of his condition should be carried out as previously outlined. If respiratory rate is increased, oxygen should be administered by nasal catheter or inhalator, preferably the latter. If cyanosis is present, the oxygen should be increased in concentrations sufficient to restore the patient's color to normal. Venesection should be performed if cyanosis and venous engorgement are evident (500 to 600 cc. of blood). However, if circulatory collapse is impending, venesection would be dangerous. To combat hemoconcentration, blood plasma should be used. Cardiac stimulants or drugs supposed to increase the blood pressure should not be prescribed nor should such drugs as morphine or the barbiturates which act on the respiratory center be administered. Chemotherapy, for the purpose of combatting a subsequent complicating pneumonia, should not be resorted to unless the patient is no longer in danger of circulatory collapse.

**Case Histories.**—An unusual type of exposure is found in the following case which came to the attention of the author.

CASE 1.—A workman was employed in general maintenance work for the Los Angeles schools. He spilled a gallon of nitric acid on a floor and wooden door. In contact with the wood, reddish-brown fumes were given off which caused him to cough. He noted no other disturbance until that night when he suffered an attack of "choking up with asthma." He stated he could hardly get his breath and breathed rapidly and that his wife noticed that he seemed blue around the mouth. The following day a physician detected signs of pulmonary irritation but did not diagnose the presence of pulmonary edema. The patient remained in bed for four days and made an uneventful recovery.

CASE 2.—This case is a report on a man who worked for a local airplane company. A leak occurred in a large nitric acid tank, the acid accumulating on the floor. The acid had eaten away a portion of the tank shell. The workman got down on his knees to investigate the leak and he attempted to stop it by wedging a piece of cast iron against the opening and then supporting this with a wood block.

Within a few minutes he coughed somewhat, but continued his attempt at repair. Ten or fifteen minutes later the plant foreman came along and immediately sensed the danger. He instructed the workman to report to the plant medical department; however, these instructions were at first ignored. In about one hour the patient noted that he was dizzy, felt sick at his stomach, and his cough had grown worse. However, he finished his shift. That night (2 A.M. in the morning), he awoke with severe chest pain, shortness of breath, and rapid respirations. His wife summoned the family physician who hospitalized the patient and immediately initiated oxygen treatment. The physician was not informed as to the nature of the man's occupational exposure.

Only routine laboratory investigations were carried out. The blood count was 5,200,000 red cells; hemoglobin, 100 per cent; white cells, 18,000; lymphocytes, 19 per cent; and polymorphonuclear neutrophiles, 81 per cent. The autopsy revealed mild cerebral edema and marked pulmonary edema.

## Ammonia

Ammonia ($NH_3$) is a nitrogen derivative. It is a colorless gas with a pungent odor. It is extremely soluble in water and gives rise to the formation of ammonium hydroxide. It is easily liquefied.

Ammonia is extensively used as a refrigerant, but there are at least thirty other occupations or processes in which ammonia is used. The aqueous solution is widely used in the various arts. Tannery and dye workers, employees working with explosives, fertilizers, sewers, varnish silk, nitric acid, etc., may be subjected to its effects.

**Signs and Symptoms.**—Conjunctivitis, swelling of the eyelids, burning of the throat, coughing, pulmonary edema, irritation of the skin, especially if it is moist, dyspnea, and respiratory failure are symptoms. Because the gas gives a warning signal, fatal dosing is not common. The maximum concentration allowable for prolonged exposure is *100 parts ammonia per million* parts of air. For a short exposure the maximum concentration allowable is from *300 to 500 parts per million* of air. Exposure to more than these amounts, or a very short exposure to from *5,000 to 10,000 parts per million*, has been fatal.

**Medicolegal Aspects.**—This gas offers little in the way of controversy. The occupation, the facts surrounding the alleged accident, the characteristic odor, and the signs and symptoms presented easily establish the case.

TEMPORARY DISABILITY.—This is usually not more than seventy-two hours. If the eyes are severely burned, there may be a temporary disability of several weeks. Pneumonia may occur following pulmonary edema or severe inflammation of the lungs. To be considered compensable, the patient must present these symptoms within a few hours or days following exposure, and not several weeks or months later.

PERMANENT DISABILITY.—This is exceedingly rare, and, if it occurs, is most likely due to involvement of one or both eyes. A concentrated amount of ammonia in contact with the cornea is likely to cause prolonged disturbance of vision or permanent damage. Once permanent loss of vision is established, the extent should be determined and so rated.

**Treatment.**—

GENERAL MEASURES.—If liquid ammonia is spilled upon the clothing, all *clothing should be immediately removed* and the body thoroughly drenched with water. The eye injured by ammonia should be immediately and copiously washed with water, and this may be followed by the introduction of a saturated solution of boric acid. If pain is severe, the use of a local anesthetic such as 0.5 per cent solution of pontocaine hydrochloride is indicated. Thereafter the application of olive oil or some similar oil is desirable. Continuous warm boric compresses to the eye may be of value. The usual treatment for corneal ulcers should be instituted if this complication occurs, and an ophthalmologist should be consulted.

RESPIRATORY AND CIRCULATORY MEASURES.—If the concentration of fumes has been severe and respiration is affected, inhalations of from 5 to 7 per cent carbon dioxide in oxygen should be given, and if pulmonary edema ensues, the use of oxygen by means of a tent or intranasal apparatus is advised. The administration of such respiratory and cardiac stimulants as the following may be of value: coramine, 1.5 cc.; metrazol, from 1½ to 4½ grains (0.1 to 0.3 Gm.); and caffeine sodium benzoate, 7½ grains (0.5 Gm.). Some of the respiratory and cardiac effects may be reflex from the pulmonary bed in origin, and because of this the intravenous administration of atropine sulfate, from 1/60 to 1/75 grain (0.0011 to 0.0009 Gm.), and papaverine hydrochloride, ½ grain (0.032 Gm.), might be of value. These should be freshly prepared from the powders just before use.

**Case History.**—The following incident of mass exposure illustrates extremely well the signs and symptoms to varying degrees of concentration[3]:

> "During an air raid in London the connecting pipe of an ammonia condenser in a brewery used as a shelter was pierced. The affected patients had mild, moderate or severe involvement of the respiratory tract. The involvement depended on the nearness of the shelterers to the pierced condenser. The symptoms of 9 mildly affected persons consisted of mild signs of catarrh limited to the conjunctivas and the upper respiratory passages, a smarting sensation in the eyes and mouth, pain on swallowing, some tightness of the throat and hoarseness. The conjunctivas were suffused, the eyelids swollen and the lips, mouth and tongue intensely reddened, dry and raw looking. The fauces presented a similar picture with the addition of edema. There was a strong smell of ammonia in the breath. The signs and symptoms of the mildly affected patients were exaggerated in the 27 who were moderately affected. There were also moist sounds in the chest, suggesting extension of the inflammatory process to the bronchi and bronchioles. In contrast to the mild cases, cough was prominent and produced tenacious and, sometimes, blood stained sputum. Auscultation revealed a poor entry of air with localized and fairly generalized ronchi. Eleven severely affected patients exhibited signs and symptoms of pulmonary edema. On admission they were shocked, restless and obviously distressed. There was slight cyanosis, intense dyspnea and a persistent coughing up of frothy sputum. The pulse was rapid and of poor volume. Generalized rales and rhonchi were audible."

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# CHAPTER XIX

# DDT (2,2-BIS-(P-CHLOROPHENYL)-1,1,1-TRICHLOROETHANE), 2,4-DICHLOROPHENOXY ACETIC ACID, AND THIOGLYCOLIC ACID AND THIOGLYCOLATES

## DDT or 2,2-bis-(p-Chlorophenyl)-1,1,1-Trichloroethane

The chemical 2,2-bis-(p-chlorophenyl)-1,1,1-trichloroethane shall hereafter be referred to only under its common name of DDT. The successful use of this insecticide by the Armed Forces led to its demand by the civilian population. It is now on the market in the form of a spray, aerosol, and powder. It constitutes no actual industrial hazard but in the author's experience several claims have been presented in which a disturbance from contact with this material has been alleged. It seems likely that the general practitioner will encounter frequent inquiry as to the toxicity of DDT, and for that reason the following opinion from Neal and von Oettingen[1] has been recorded here.

DDT is a white, crystalline powder, insoluble in water but soluble in oils and certain organic solvents.[1] It is tasteless and, when pure, practically without odor. When DDT is applied in solutions, or as a powder, it adheres to any surface with which it comes in contact, and for this reason it *is not very liable to form a dust* upon agitation of the air. The development of DDT as an insecticide has been accompanied by changes in the methods of application of insecticides, inasmuch as it has been found necessary to decrease the particle size of the mist, spray, aerosol, and smoke to relatively small particles or droplets. Since absorption through the respiratory tract and penetration into the alveoli depend largely upon the size of the particles inhaled, the use of aerosols, smokes, and power sprayers produces particles which can be absorbed through the lower sections of the respiratory tract. Because of the relatively small amount of DDT present in insecticidal mixtures necessary to produce the desired insecticidal effect, the actual concentration of DDT in the air remains relatively small.

DDT has been subjected to intensive investigation because of its widespread utility and especially because governmental experts knew that it would be used widely by the Armed Forces. Accordingly, before it was released, experimental and human studies were conducted by the United States Public Health Service at the Industrial Hygiene Research Laboratory, National Institute of Health, Bethesda, Maryland, under the supervision of Dr. Paul A. Neal,[2] Senior Surgeon. These tests were carried out mostly on the powder. In 1943, a study of the toxicity of DDT when used as an aerosol (vapor mist), as a powder, and as a spray in deobase was undertaken at the request of the Surgeon General's Office of the United States Army. All the aspects involved in the use of DDT were discussed on May 7, 1943, at the National Institute of Health, with Lt. Col. W. S. Stone of the Surgeon General's Office, United States Army; Dr. F. C. Bishopp and Dr. H. H. Stage, of the Bureau of Entomology and Plant Quarantine, United States Department of Agriculture; Prof. P. A. Buxton, of the British Scientific Committee; Dr. H. O. Calvery, of the Food and Drug Administration; Dr. R. E. Dyes, Director of the National Institute of Health; and Dr. M. I. Smith, of the Division of Physiology, Dr. W. H. Wright, of the Zoology Laboratory, and Dr. P. A. Neal and Dr. W. F. von Oettingen, of the Industrial Hygiene Research Laboratory of the National Institute of Health. At that meeting, after reviewing the importance of DDT as an insecticide, it was decided that the Industrial Hygiene Research Laboratory should study the toxicity and potential danger resulting from the inhalation of DDT as an aerosol, dusting powder, and mist, thus supplementing the toxicologic work of Dr. H. O. Calvery and Dr. M. I. Smith, which at that time was already in progress. It was further decided that monthly progress reports should be prepared by the different groups and forwarded to all persons engaged in this study as well as to the National Research Council. This exchange was extended to Dr. R. A. Kehoe, of the Kettering Laboratory, who was also investigating the toxicity of this material.

The materials used in this study were prepared and supplied by the Bureau of Entomology and Plant Quarantine of the United States Department of Agriculture. The research was begun in the middle of May, 1943, and was completed by the end of September of the same year. The final report was submitted on September 28, 1943, to the Fifth Conference on Insecticides and Insect Repellents, Committee of Medical Research, National Research Council; the Surgeon General's Office of the United States Army; and the Bureau of Entomology and Plant Quarantine of the United States Department of Agriculture.

The acute toxicity of various DDT aerosols was studied in mice. rats, guinea pigs, and dogs.

Two human beings (aged 42 and 54 years) were subjected to an intermittent exposure of 0.035 mg. DDT per liter (1 mg. per 1,000 cubic feet) of air, for one hour daily, during a period of six days. In these experiments, 10.4 mg. of DDT aerosol, consisting of 5 per cent DDT, 10 per cent cyclohexanone, and 85 per cent Freon, were introduced every fifteen minutes into a hermetically sealed chamber of 14,750 liters capacity. The air in the chamber was agitated by a small electric fan placed in one corner. The subjects underwent medical examination before entering the chamber, including the determination of the pulse rate, blood pressure, respiratory rate, size of pupils and appearance of eyegrounds and conjunctivae and presence of nystagmus, inspection of the throat, steadiness tests (finger-nose test and steadiness of extended fingers), and biceps and Achilles reflexes. The urine was tested for albumin, sugar, urobilin, urobilinogen, cellular constituents, and casts, and its pH and specific gravity were determined. Before the beginning of the experiment and at the end of the exposure period the blood picture was studied with regard to red blood cells, hemoglobin, white blood cells, and differentials. Blood samples drawn before and after the exposure were studied with regard to icterus index and the cephalin-cholesterol flocculation test. A set of psychophysiologic tests, including a mental alertness test, were made before and after each exposure.

No subjective systemic manifestations of DDT intoxication were noted. The results of the physical examination were negative and no significant physiologic aberrations were noted other than a drop in the hemoglobin and red blood cell count. It will be noted later that even following a more severe exposure test the values before and after exposure remained the same. The urinary findings were essentially negative except for the fact that on three occasions Subject A showed a 1 plus test for urobilinogen which was quite faint in a dilution of 1:20; the significance of this finding is very doubtful because the icterus index of this individual was only 3.0. The cephalin cholesterol flocculation test for liver damage was negative when applied to these two test subjects.

Under the conditions of this experiment the concentration of DDT in air was supposed to be, according to a weight-volume ratio, 1 Gm. per 1,000 cubic feet, or 0.035 mg. per liter, so that the volume of air (approximately 600 liters) inhaled by each test subject during the exposure would contain a total of 22.8 mg. of DDT. It was found, however, that only 3.1 to 4.0 mg. of DDT could be determined in 634 liters of air by chemical analysis.

It appears, therefore, that even with the spraying of DDT aerosol every fifteen minutes, the material settles out rapidly and the actual concentration of DDT in air is considerably below the amount calculated. Furthermore, the experiment shows that inhalation of 3.1 to 4.0 mg. of DDT in the form of DDT aerosol for a period of one hour daily, on six consecutive days, causes no subjective or objective symptoms in human subjects.

In a second series of experiments the exposure was intensified by introducing 10.4 Gm. of the same DDT aerosol into the exposure chamber at intervals of five minutes instead of at intervals of fifteen minutes. Four weeks after termination of the first experiment, both subjects were exposed to this more severe concentration. They underwent careful medical examination before and after the exposure period. However, no clinical signs of any toxic effects were revealed. Both subjects suffered a slight irritation of the upper respiratory tract and of the eyes, this effect becoming noticeable from five to ten minutes after the beginning of the exposure, and increasing thereafter to a moderate degree. It should, however, be pointed out that in both subjects the vibrissae in the nostrils were covered with a heavy, white deposit of DDT, indicating that a large portion of the material inhaled was filtered off before entering the respiratory tract. One subject was stripped to the waist during the exposure and the arms and shoulders of the other were exposed. The hands and forearms of one subject were coated with DDT during the entire experiment. This was caused by the escape of some of the aerosol during transfer from a large cylinder into a small receptacle. Neither of the two subjects showed any aftereffects and all biochemical tests performed with the blood and urine failed to show any significant deviation from the normal.

These experiments revealed, therefore, that inhalation for one hour daily, on five consecutive days, of a DDT aerosol produced by dispensing 10.4 Gm. of the aerosol containing 5 per cent DDT, 10 per cent cyclohexanone, and 85 per cent Freon into a hermetically sealed chamber of 14,750 liters capacity, corresponding to a total of 124.8 Gm. of the aerosol, produces no toxic effects in human subjects.

Dr. Paul Neal, who directed much of the research on DDT, stated in 1946[3] that:

"In view of the extensive use of DDT as an insecticide, well over one million humans have been exposed to this substance. It is obvious that some of these will develop diseases the true character of which is not readily diagnosed, and therefore easily credited to the exposure to DDT."

In summary, says Dr. Neal, it can be stated that DDT has not been found to be toxic in human beings, and other than irritation of the skin in individual workers in the manufacturing of this substance, no ill effects have been noted.

In a later paper, Neal and von Oettingen[4] reviewed previous investigations and concluded:

> "The health hazards associated with the use of DDT as an insecticidal agent may be divided into two types:
>
> "(1) The hazards connected with the manufacture, packaging, mixing and handling of DDT-containing insecticides, as well as the hazards connected with the applications of such insecticides, both as pertains to their use by trained sanitarians, as well as their household application.
>
> "(2) The dangers to the general population resulting from the ingestion of various types of foods contaminated with small quantities of DDT.
>
> "With regard to type of hazard number 1, the dangers incident to its manufacture, mixing and application can be controlled by employing certain commonsense precautions. These precautions which have been defined by various governmental agencies have proven to be satisfactory in that, as far as can be determined at this time, no cases of DDT poisoning *per se* have been proven to occur among the some millions of individuals who have been potentially exposed.

"The second type of hazard, contamination of food with DDT or what is the maximum allowable tolerance of DDT residue on foods, cannot be definitely answered at this time on the information available, in our opinion. It seems obvious, however, that the use of DDT insecticides for certain underground crops, such as potatoes and peanuts, would offer practically no hazard. This question is at present the center of interest of different governmental agencies who are working in closest collaboration to solve it.

"Although DDT is deadly to many insects, it is not harmful to man if properly applied. DDT is definitely less toxic than Paris green and sodium fluoride—long in use as common insecticides. But as with any insecticide, there are certain precautions which must be taken to avoid any possible harmful effects.

"In spite of its toxicity, no harmful effects have been observed in animals exposed in rooms containing many times the recommended insecticidal concentration of DDT in sprays or dusts. But careless use and exposure to abnormally high concentrations of DDT may cause toxic effects. Therefore, it is essential to follow carefully the directions for its use as a household insecticide.

"Since there is no specific antidote for DDT poisoning, the treatment has to be symptomatic. It is of paramount importance to prevent further absorption by removing any contamination from the skin by washing with soap and warm water. Following accidental ingestion of DDT, it should be removed promptly from the stomach by gastric lavage with warm water, or from the intestine by saline cathartics. Castor oil or other fatty cathartics or emulsions should never be used. Animal experiments indicate that nervous manifestations, such as tremors and convulsions, can be controlled by phenobarbital. In severe cases sedation is especially indicated and may prove of life-saving value.

"It is very unlikely that food contaminated with DDT from ordinary home use will cause toxic effects in man, but such contamination should be avoided by removing food from the room or covering it during spraying.

"DDT in *dust form* is not absorbed through the skin unless greases, oils, or greasy skin lotions are already present on the skin. Nevertheless, DDT powders should not be allowed to remain on the skin, and excessive inhalation of the powder should be avoided. Since the diluted dust (if uncolored) may be mistaken for flour or other foodstuffs, the material should be carefully labeled and every precaution taken to keep it away from children. Any danger of food contamination should be avoided.

"DDT in *oil solution* is readily absorbed through the intestine and is also absorbed through the skin. Therefore, DDT-oil solutions should not be allowed to remain on the skin or saturate clothing. Wash the hands and exposed skin with warm soapy water; and if oil solutions or concentrates are spilled on the clothes, change them promptly. Avoid inhaling the mist and contaminating food with the spray. *Never use it on the skin or coat of animals.* If the solvent is inflammable, don't use it near a fire.

"It should be pointed out that many of the solvents (kerosene, etc.), used in preparing DDT insecticides in themselves may cause irritation of the skin and other harmful effects when handled carelessly. By observing proper precautions and cleanliness, these can be avoided.

"If a good deal of spraying is to be done it is advisable to wear gloves, and a respirator to avoid excessive contact and inhalation of DDT and its solvents."

## 2,4-Dichlorophenoxy Acetic Acid

The chemical 2,4-dichlorophenoxy acetic acid (referred to commonly as 2,4-D) is finding increasing use in the destruction of weeds—a "weedicide." Because of its power to kill weeds, this substance will undoubtedly be suspected of being injurious to human beings, an error similar to that made with respect to DDT. Because of such claims, various authentic sources were approached in order to determine the toxic properties of 2,4-dichlorophenoxy acetic acid. From the United States Department of Agriculture, from personal correspondence with Dr. Paul A. Neal [5] and Dr. D. D. Irish,[6] of the Dow Chemical Company, and from several scientific journals mentioned in the appended reference the following facts were ascertained.

The United States Department of Agriculture did not hesitate to recommend the formula 2,4-D, which is the basic component of Dow's A-510, as a substance completely inoffensive to human beings and animals. This same Department experimented on the human being by administering small doses of 2,4-D daily, during a considerable period of time. No evidence of any untoward effects was noted. Experimental ingestion of larger doses over a shorter period of time were equally tolerated without the individual experiencing any disturbance.

The United States Department of Agriculture also made heavy applications of this chemical to certain pastures, and then permitted sheep and goats to browse where it had been spread. In feeding, these animals showed no preference or dislike for the places where the formula had been applied. Likewise, food in which 2,4-D had been incorporated did not affect goats that fed upon it.

The Department also published the report of a case in which a cow was fed daily over a long period of time with greens which contained relatively heavy doses of pure 2,4-D. It was pointed out that no effects from the feeding with 2,4-D were observed in the cow or in the calf which was born during the period in which the experiments were carried to completion. Traces of 2,4-D were found in the blood of the cow, indicating that its organism was normally eliminating the chemical product. After some time, the cow was slaughtered and several experts in the animal industry conducted an autopsy, carrying out careful chemical analyses of all the organs of the cow, without being able to find a trace of the chemical compound and without perceiving any effect of the ingestion of large doses of 2,4-D.

The United States Department of Agriculture also advised that the manufacturers of 2,4-D had encountered no difficulty in the production of the compound with any of their employees. It should be remembered that those who are engaged in the actual manufacturing of 2,4-D are in continual contact with concentrated forms of the substance. For this reason, it was not considered likely that men who are employed in agricultural fields applying the chemical substances in a highly diluted form should suffer any adverse effects to their health. The manufacturers reported a case in which a man in the State of California, working extensively with 2,4-D, had broken out in an eruption of the skin, but they attributed it to other causes, and not to 2,4-D. According to the report the eruptions had disappeared before it was possible to make a study of them with the purpose of determining their cause.

An additional report from the Department of Agriculture, and related by Mitchell and co-workers[7] in the *Journal of Animal Science,* records the following:

> 2,4-D spraying of pastures to kill weeds has not proved injurious to cows or sheep grazing on the treated vegetation, according to results of tests by the United States Department of Agriculture's Research Administration. The experimenters found no indication that any of this herbicide, known chemically as 2,4-dichlorophenoxy acetic acid, passed from the digested herbage into the cow's milk. Even when they added the pure chemical to the grain ration of a cow at the rate of 5.5 Gm. a day for more than 100 days she showed no ill effects in weight, production of milk or even in appetite.

Some of the chemical fed in the ration showed in tests of the blood serum, but there were no indications of it in any other parts of the body. A calf fed on the milk of a cow receiving the 2,4-D developed normally and the chemical could not be found in its blood serum.

The studies were carried on jointly by scientists of the Bureau of Dairy Industry and the Bureau of Plant Industry, Soils and Agricultural Engineering. Although they found that 2,4-D mixed with a commonly used spray spreader called Carbowax is not toxic to sheep and cows, they caution that their conclusions do not cover proprietary mixtures of 2,4-D and other materials that might possibly be bad for animals. Probably the most interesting information regarding the nontoxic effects of 2,4-dichlorophenoxy acetic acid is to be found in the suit which the Ambler, Pennsylvania, township brought against the manufacturer of this chemical. The people of this community brought suit because the chemical escaped into the air and killed the plant life for some miles around. But the suit contained no complaint to the effect that human beings were irritated or harmed. It was found that the chemical had a selective action upon plants but not upon human beings. In an account of this damage suit it is stated[8]:

> "An interesting phenomenon concerned with the absorption of phenollike vapors by plants was observed during the summer of 1945 in and about Ambler, Pennsylvania. A factory located on the outskirts of Ambler started the commercial production of *2, 4-dichlorophenoxy acetic acid (2, 4-D).* This product has been used as a plant growth regulator and has been developed by the Bureau of Plant Industry, Soils and Agricultural Engineering, as a weed killer.
>
> "In the production or purification of the product at Ambler, phenolic vapors escaped into the atmosphere and were distributed over several square miles of the area surrounding the factory. The concentration of the vapors varied with the atmospheric conditions; on clear days the concentration was slight or not

noticeable; on cloudy days with low atmospheric ceilings the vapors were quite noticeable at distances of one to two miles from the factory. The summer in this vicinity was a wet one, the number of cloudy days and nights being above average.

"One determination of the concentration of the vapor taken by the Pennsylvania Department of Health at the source is reported equivalent to parts of dichlorophenol at 4 p.p.m. However, at this concentration near the source, the odor is quite noticeable. While the vapors may be objectionable, they have not yet been regarded as a human health hazard, the factory workers having been exposed to these concentrations without any reported ill effects."

## Thioglycolic Acid and Thioglycolates*

Thioglycolic acid, $HS.CH_2COOH$ (mercaptoacetic acid), is a colorless liquid with a strong, skunklike odor. Hydrogen sulfide is one of its chief products of decomposition.

In 1941, Bunce, Parker, and Lewis[9] reported a death which they attributed to the absorption of toxic material from a permanent hair wave solution. Cotter, in 1946,[10] suggested the possibility of systemic disturbance from hydrogen sulfide existing in a permanent wave solution. In December of 1946, McCord[11] investigated the toxicity of numerous (and possibly all) permanent wave solutions on the market.

The beauty parlor would seem to be more than a medical stone's throw from the office of the industrial physician, yet it is estimated 55,000,000 permanent waves are given to the female population annually. Since the beautician is exposed to the substances contained in thioglycolic acid which may be toxic under given situations, she is a subject to be protected by the industrial hygienist. It appears that the most toxic agent within the preparations used for permanent wave fixation is hydrogen sulfide. In this regard we quote McCord[9]:

"The toxicity of hydrogen sulphide is such that the standard for prolonged exposure devised by the American Standards Association is 20 p.p.m. for standard conditions. If it may be assumed that every adult breathes approximately 1 meter of air per hour, it follows that at this tolerated level of 20 p.p.m. of air, 27.8 mg. of hydrogen sulphide entering the body still falls within the safe limit. If the permanent waving process does not extend beyond 4 hours, which appears ample, 111.2 mg. of hydrogen sulphide might be tolerated. Therefore, the question involved is whether under any ordinary circumstances of permanent waving, those exposed might have imposed on them hydrogen sulphide in excess of 111.2 mg. for customers or 222.4 mg. for the operator working an 8-hour day.

"Commercial thioglycolic acid (about 84%), standing in half-filled bottles, may give rise to some 200 p.p.m. of hydrogen sulphide within the bottle. Should this be removed, as much as 10 p.p.m. may reappear in 10 minutes, in the superimposed air, all tests being made with the silver cyanide method employing hypersensitive tubes.

"In the case of thioglycolates in the form of commercial hair waving preparations, similar tests for hydrogen sulphide indicate concentration from

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* This discussion actually belongs in the chapter on Hydrogen Sulfide, but decision to include it in this book came after the aforementioned chapter was written.

2 to 20 p.p.m. in the air above the liquid levels in containers. It follows as possible that large evaporating surfaces, such as provided by the hair of the head, or heat, or air oxidation, or chemicals of the hair, might so act as to accelerate this decomposition to dangerous levels of hydrogen sulphide."

McCord summarizes his study as follows:

"The physiologic properties of thioglycolic acid and thioglycolates have been investigated, foremostly in relation to cosmetic use. This report is concerned with the chemistry and physics of hair waving, primary skin irritation, the prospects of sensitization, the extent of instability of thioglycolic acid and thioglycolates, stabilization, systemic action after subcutaneous introduction, and direct action on the hair.

"The conclusions reached are:

"Thioglycolic acid in relatively low concentrations is toxic and may induce necrosis from subcutaneous implantation.

"Sodium thioglycolate when free from thioglycolic acid, in concentration approximating 4%, in contact with the skin for 4, 6, 9, 24, 48, 72, and 96 hours has not produced demonstrable damage.

"Ammonium thioglycolate, commercial preparations of unknown concentration, but designed for identical application, induced skin injury (7.4% frequency) at the 24 hour contact period but not for 4, 6 or 9-hour periods.

"Sodium thioglycolate in concentrations approximating 7% provoked skin irritation at and above the 24 hour contact period.

"These conclusions derive from 1129 patch tests applied to humans.

"The allergenic properties of both thioglycolic acid and thioglycolates as measured by repeated patch tests were appraised as low or nonexistent.

"The total quantity of hydrogen sulphide arising after induced decomposition of either thioglycolic acid or thioglycolates is insufficient to produce systemic poisoning.

"Stabilization of thioglycolic acid and thioglycolates to prevent the appearance of hydrogen sulphide (or not more than mere trace) is practical.

"Thioglycolates exert some action on hair tensile strength, elasticity, and cellular state, but the degree of such does not preclude use.

"Continuing exposure for professional beauty shop operators, to thioglycolates and other cosmetic chemicals requires a program of protection, unnecessary in occasional household use."

As previously pointed out, there are occasions when unforeseen dire results may occur to the customer obtaining a permanent wave, but it is the beautician who needs the protective measures most. These as recommended by McCord are:

"For the purpose of safeguarding the persons most exposed to thioglycolates along with divers substances possibly more harmful, a number of recommendations are now introduced:

"1. The product or products selected for use should be limited to those well established as standardized and not subject to possible mishaps or haphazard formulation.

"2. All operators should wear surgical gloves in connection with the application of various beauty shop chemicals, including thioglycolates, neutralizers, fixatives, etc.

"3. Since some cold wave preparations retain offensive mercaptan odors to the point of discomfort but not damage to health, it may be necessary that exhausts be installed for air dilution.

"4. Beauty shop operators with skin diseases on exposed areas particularly should avoid contact with various substances common to beauty shops, and including thioglycolates, neutralizers, fixatives, etc.

"5. In the presence of skin diseases with characteristics suggesting contact dermatitis or sensitization, patch tests should be carried out by a physician in order to determine the exact agent responsible. This is a general statement and without specific reference to cold wave preparations. After the establishment of the precise cause of such affection avoidance of direct contact with that substance may be sufficient to escape perpetuation of the skin condition, but on rare occasion proximity of the offending substance from use by others may prove to be injurious.

"6. While it is likely that thioglycolic acid, as such, will not be introduced into beauty shops, it should be recognized that this agent is distinctly harmful, with prospective damage increasing with the percentage of the thioglycolic acid.

"7. Since the work of beauty shop operators necessitates almost endless washing of hands and arms, and equally frequent contact with water in connection with customer needs, all operators should make use of frequent applications of water-impervious creams, although this may not be necessary if surgical gloves are worn."

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# PART THREE
# THE METALS

## CHAPTER XX

## LEAD

The diagnosis of lead poisoning is too frequently assumed and too rarely proved. Lead poisoning is one of the oldest of all occupational diseases. The passing years have taught us its clinical syndrome and investigators have provided the laboratory basis for accurate diagnosis. Inaccuracy of diagnosis in cases of alleged lead intoxication stems from the physician's failure to carry his investigations sufficiently far. It is a fact that *the one laboratory test* relied upon by the majority of physicians is the "stipple cell count." Yet such a test does not vouchsafe a correct diagnosis. The fault lies with the textbooks of medicine used in our medical schools, which invariably state that the diagnosis of lead poisoning is based upon the "symptoms of intestinal colic and constipation; the finding of a lead (blue) line on the gums and the presence of basophilic cells in the blood smear." As subsequent discussion will show, this is an insufficient basis for diagnosis.

**Occupational Exposure.**—The occupations which involve the use of lead are too numerous to mention. Those which offer the greatest hazard are smelting, refining, and reclaiming of lead, as well as enameling, glazing and painting; the application of heat to surfaces containing lead or giving rise to lead oxides, as in welding or burning, is also liable to produce lead poisoning. A high incidence of lead poisoning occurs in the small plants without proper medical supervision which make electric batteries.

**Absorption and Excretion of Lead.**—Inhalation of lead dust or fumes is the chief source of intoxications. The next most common route is by ingestion, while cutaneous absorption is not likely. The question of absorption of lead from bullets or foreign bodies located in the soft tissues is of no industrial importance. It is, however, believed that such absorption can occur, but according to Machle[1] it is a rare incident.

Various theories have been advanced as to how lead is absorbed once it enters the respiratory tract. Some believe that it is accomplished in part by phagocytosis and in part by a breakdown of the particles into a fine diffusion which is then absorbed by the moist epithelium. Some hold that lead compounds are converted to lead carbonate by the action of $CO_2$. To the average reader the mechanism of absorption is relatively unimportant, but it should be appreciated that lead dust does not penetrate the lung to cause appreciable fibrosis, which may be the action of certain other dusts. Once lead is absorbed by way of the respiratory tract it enters the blood stream, from whence it is dispersed into the tissues where it is accumulated.

Lead which enters the intestinal tract is passed out in the feces, but only small amounts are absorbed. Kehoe and co-workers'[2] experiments on human beings indicate that the absorption, even of soluble lead, from the alimentary tract, is slow and ineffectual. "Calculated on the most liberal basis only about one-tenth of that administered was absorbed." The small amount of lead which is absorbed from the intestinal tract, probably by way of the lymphatics, enters the portal circulation, passes to the liver, and may then be returned into the bowel without ever reaching the systemic circulation. A portion may be stored for a time in the liver and enter the systemic circulation. Gant[3] claims that an equivalent amount of lead is 10 to 100 times more toxic when inhaled than when ingested.

**The Fate of Lead in the Body.**—Once lead has been absorbed it is distributed to the liver, the spleen, the kidneys, the heart, the lungs, the brain, the muscles, and the skeletal system. Analysis of the tissues of all adult human beings (North Americans) will reveal some lead in these organs, even though they have had no occupational exposure. An approximate range of normal values is given in Table X.

TABLE X

| | LIVER | SPLEEN | KIDNEYS | HEART | LUNG |
|---|---|---|---|---|---|
| Minimum | 0.04 | 0.01 | 0.015 | 0.01– | 0.01 |
| Maximum | 0.28 | 0.07 | 0.16 | 0.07 | 0.06 |
| Arith. mean | 0.12 | 0.03 | 0.05 | 0.03 | 0.03 |
| | BRAIN | MUSCLE | FLAT BONE | LONG BONE | |
| Minimum | 0.01 | 0.01– | 0.21 | 0.67 | |
| Maximum | 0.09 | 0.17 | 1.11 | 3.59 | |
| Arith. mean | 0.04 | 0.03 | 0.65 | 1.78 | |

Lead which has been absorbed in excess of the normal amount is fixed in the soft tissues for a brief time, after which it is removed from these organs to enter the general circulation and then to be excreted.

The degree to which lead is fixed in the skeletal system, however, is a controversial point. Aub and associates[4] pioneered the belief that lead is transported as a finely divided colloidal lead phosphate to be deposited as a tertiary lead phosphate in the bones. According to these authors, once deposited, the lead metabolism closely parallels the action of calcium. Following his original work, Aub, along with Robb and Rossmeisl,[5] contended that most of the lead in the bones is located in the trabeculae, and not in the cortex. Since the trabeculae act as a storehouse for available calcium, it was believed that lead similarly stored is available and more easily mobilized.

The leading authorities in the field are not unanimous in their belief that lead is fixed in stable form in the skeletal system. In a personal communication from Kehoe,[6] that author writes:

> "I would say in relation to this whole question of fixation of lead in the body that our experience and observations will not permit us to accept the hypothesis that lead can be bound in stable form in any part of the body, with the possible exception of the nerve tissues. The idea that lead is fixed in stable form in the skeleton either normally or through any kind of therapy is, in our opinion, untenable. In fact, all of our evidence tends to show that if the lead content of the body is elevated above the normal, the excretion of lead in the urine will be more or less correspondingly elevated. The role of calcium and phosphorus in relation to lead metabolism has not been worked out. We have never found any evidence that such therapy produces more than minor changes in the distribution of lead in the body and in the rate of excretion from the body. We do not on this account condemn the use of calcium or phosphorus, or both, in the treatment of lead poisoning, but we do, however, believe that whatever benefit is achieved is not the result of mobilization or demobilization of lead in relation to depots of deposit."

The question whether lead can be stored in the bones and subsequently released from this storehouse is of more than academic interest. Those who believe that lead can be stored and later released have devised their therapy accordingly, while those who disbelieve this theory refrain from making any attempt to influence the fate of lead in the body.

**Pathology.**—The most consistently harmful effect of abnormal lead absorption is that to the blood cells. Anemia, although not usually severe, is invariably present. Lead acts upon the surface of the red blood cells in the peripheral circulation, causing them to become brittle and to alter their size and shape. Because of this brittleness they are easily destroyed by trauma to the circulation. With increase to hemolysis there occurs an increased hematoporphyrinuria. Evidence of alteration of the derivatives of hemoglobin has also been observed, such as increased deposits of hemosiderin in the tissues, hyperbilirubinemia, increased bilirubin in bile and urine, and increased excretion of porphyrine. There is usually no change in the leucocyte count and it would be unadvised to ascribe any slight variation from the normal to lead absorption.

When destruction of red cells in the circulation occurs, there takes place a compensatory stimulation to the bone marrow, resulting in an increase of the immature erythrocytes and reticulocytes. As aptly described by McCord and associates[7]:

> "In normal adult human life, the content of erythrocytes in the blood stream is maintained on a fairly uniform level by the orderly entry of new cells from the bone marrow replacing those that have been destroyed.

> "These new cells are essentially mature, only about 1 per cent exhibiting any of the several known characteristics of immaturity. In bone marrow, during the formative period of erythrocytes, an entirely different cytology takes place varying with the stage of development of the red cells. Readily there may be demonstrated nuclei, protoplasm, and basophilic substance. In due time a change not known for any other cells of the body occurs; a chemical (hemoglobin) replaces the protoplasm and the nucleus is extruded. The erythrocyte is then ready for its chief function in the circulatory blood. The mechanism which liberates cells when mature and conversely retains undeveloped cells is not known. However, the functioning of such a mechanism is well established.
>
> "Under conditions in which toxic agents exert an action on bone marrow, and under other conditions in which physiologic demands are made, increased numbers of erythrocytes enter the blood stream. As examples of the former may be cited lead, benzol, toluol, xylol, and possibly arsenic and chlorinated hydrocarbons, such as carbon tetrachloride; of the latter, the effects of high altitudes constitute an obvious instance. The chief characteristic of these liberated cells is the presence of basophilic substance."

Of particular interest in the blood studies carried out on individuals suffering from lead poisoning is the presence of these punctate basophilic cells. Basophilic stippling is always present in the acute stage of lead intoxication, but *it is not pathognomonic of plumbism.* Stippled cells may be present in pernicious anemia, secondary anemia, leucemia, hemolytic jaundice, neoplasms, or in toxic states due to the chemicals previously mentioned (Fig. 29).

The mechanism which produces intestinal colic in lead poisoning is not clearly understood. The pain is obviously the result of constriction and spasms of the intestines. Increased peristalsis, enormous dilatation of the cecum and colon, contraction rings in the large bowel with delayed emptying time of its content have all been observed. At one time it was believed that lead had a selective action upon the nervous structures of the gastrointestinal tract, but more recently this idea has been abandoned. It has been pointed out that neither atropine nor epinephrine has any influence upon the peristalsis or contractions of the intestines.

Various investigators have now come to the conclusion that lead acts directly upon the smooth muscle fibers. However, they offer no satisfactory explanation of this action. Minot[8] suggested the possibility that lead intoxication might perhaps be accompanied by an increased permeability of the muscle cells to certain inorganic ions and to changes in electrolyte equilibrium.

In the earlier literature on plumbism, references are made to such pathologic changes in the intestinal tract as gastritis, minute hemorrhages, necrosis of the gastric epithelium, and other pathologic conditions. More recent authorities, however, have not noted such changes.

Wrist drop and more rarely ankle drop may result from prolonged exposure to lead, but neither of these conditions is seen very often these

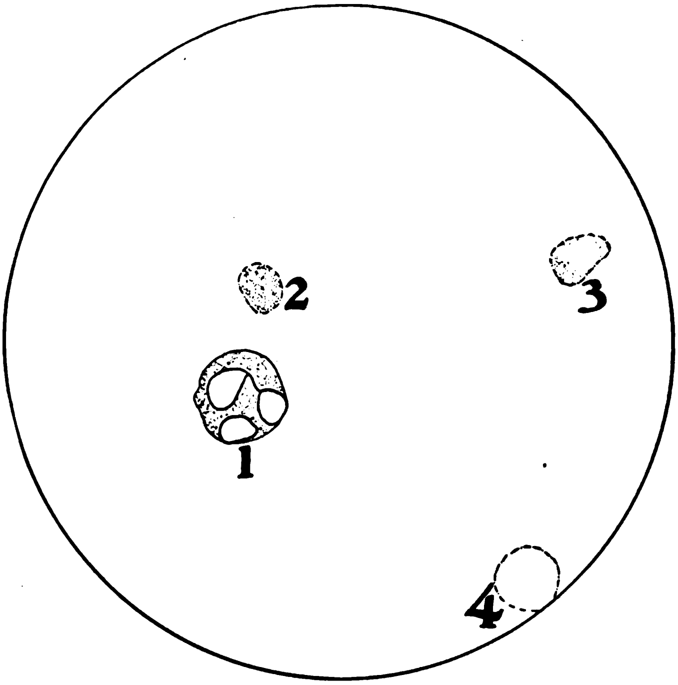

Fig. 29.—Anemia of lead poisoning (secondary anemia).

1. Neutrophil.
2. Erythrocyte with coarse basophilic stippling.
3. Erythrocyte with fine basophilic stippling.
4. Polychromatocyte.

**Blood Findings:**

| | |
|---|---|
| Hemoglobin | 6.5 Gm. (Newcomer's method). |
| R.B.C. | 3,150,000 per c.mm. |
| W.B.C. | 8,450 per c.mm. |
| Platelets | 295,000 per c.mm. |
| Color index | 0.6 |
| Volume index | 0.8 |

**Differential:**

| | |
|---|---|
| Myelocytes | 0% |
| Juveniles | 5% |
| Bands | 11% |
| Segmenters | 60% |
| Total neutrophils | 76% |
| Lymphocytes | 16% |
| Eosinophils | 2% |
| Monocytes | 6% |

Erythrocytes—hypochromic and microcytic with pronounced basophilic stippling.
(From Kracke: Diseases of the Blood, Philadelphia, J. B. Lippincott Company.)

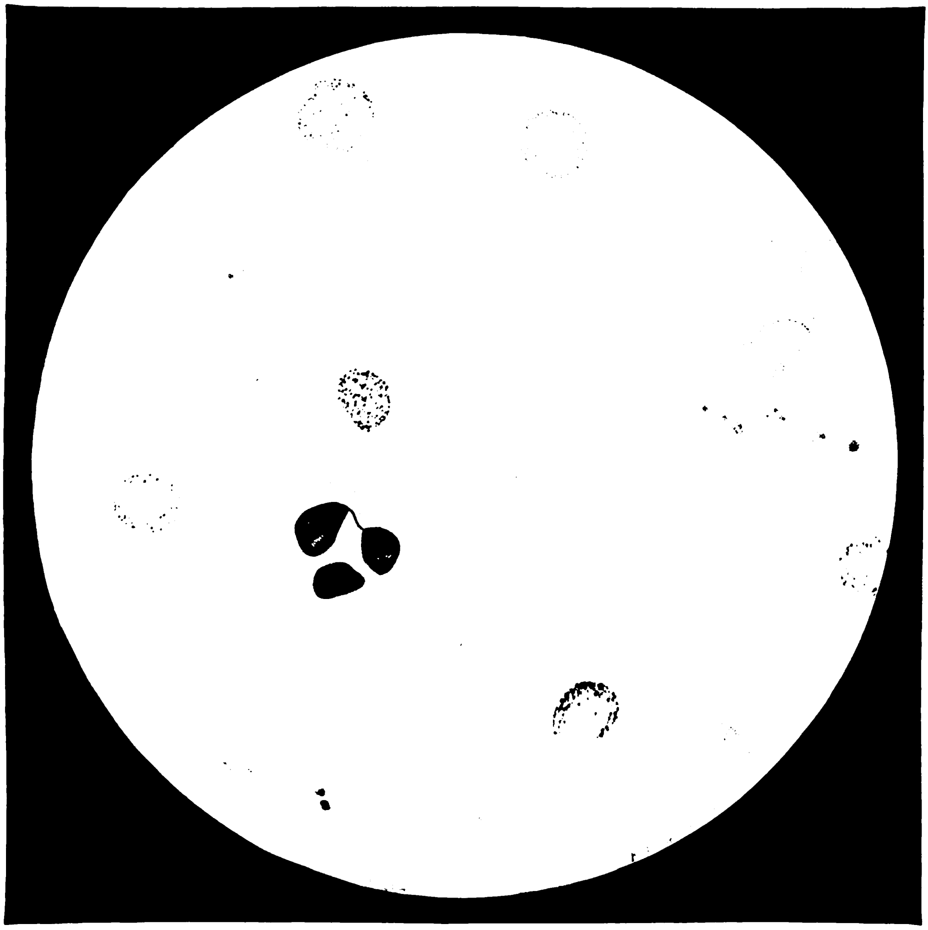

Fig. 29.
*(For legend, see opposite page.)*

days, due to the existence of better control methods in industry. Again, the explanation for this phenomenon is not specific, and considerable controversy is carried on as to its specific cause. Some investigators feel that the pathology resides in the peripheral nerve supply, some have attributed it to morphologic changes in the anterior horn cells and lateral columns of the spinal cord, and still others state that lead acts directly upon the involved muscles.

It is generally recognized that extensor weakness precedes the appearance of wrist drop or palsy. It has also been noted that the muscles affected are those which are used most frequently, and which are therefore most readily fatigued. Recently, the opinion has been brought forth that a physiologic reaction within the involved muscle groups is accountable for the interference with normal muscle contractions. The following various viewpoints on this chemical phenomenon have been collected from the literature by Cantarow and Trumper.[9]

"It was suggested by Hitzig that as a result of the greatly increased circulation in these muscles they were exposed to disproportionately large quantities of lead. However, as stated by Aub, this does not explain the mechanism of action of lead on the muscles. Some early investigators of this problem found that muscle exposed to lead rapidly became fatigued and inexcitable, and that its relaxation time was prolonged. That this effect was due to some change in the contractile elements in the cells was suggested by the fact that it occurred on direct stimulation of the muscle. This view is also supported by the observation by Reznikoff and Aub [10] that action potentials could be obtained from the nerve but not from the muscle of a nerve-muscle preparation exposed to lead. The studies of Reznikoff and Aub on isolated muscle showed that exposure to lead results in more rapid fatigue and greater difficulty in recovery. They concluded that lead interferes markedly with the function of isolated muscle. These authors made the interesting and important observation that after exposure to lead there was a marked increase in the rate of diffusion of inorganic phosphate from the muscle, a phenomenon which they attributed to alteration of the surface membrane with increase in its permeability. They believed that this increased permeability is accomplished in much the same way as in the case of the red blood cells, being due essentially to precipitation of insoluble lead phosphate at the surface of the cell with liberation of free acid. According to this hypothesis, excessively large quantities of lactic acid, which diffuse from fatigued muscle cells in regions of muscular activity, convert the circulating lead phosphate into lead lactate. The soluble lead lactate comes in contact with inorganic phosphate at the surface of the muscle cell, lead being precipitated as the insoluble tertiary phosphate with the liberation of free lactic acid.

"Subsequent observations regarding the physiology of muscular contraction necessitated reinterpretation of these findings. The following are of significance in this connection: (a) the discovery of phosphocreatine and the demonstration that inorganic phosphate constituted only 15-20 per cent of the total acid-soluble phosphorus of the muscle; (b) the demonstration of the fact that energy for muscular contraction is supplied by the breakdown of phosphocreatine into creatine and inorganic phosphate; (c) the observation that phosphocreatine cannot diffuse through the muscle membrane whereas creatine and phosphate are freely diffusible. In view of the fact that the inorganic phosphate content of muscle is now known to be only 20-25 mg. per cent, and in view of the observation that the diffusibility of inorganic phosphate is the same for resting and fatigued muscle, Steiman [11] raised the question as to whether the great increase in the diffusion of phosphate from 'leaded' muscle might not be due to hydrolysis of phosphocreatine with consequent increase in the concentration of inorganic phosphate in the muscle. He found that the resting level of phosphocreatine in the muscles of animals exposed to lead was much lower than normal, while the inorganic phosphate was greatly increased. Whereas in normal muscle the phosphocreatine hydrolyzed during a two-minute tetanus was completely resynthesized after a half-hour recovery period, practically no resynthesis occurred in lead poisoned muscle. The loss of inorganic phosphate from the latter was shown to be due to the increased amount of inorganic phosphate resulting from phosphocreatine hydrolysis. Since there is a direct correlation between the presence of phosphocreatine and the ability of the muscle to contract, the poisoned muscles with a low phosphocreatine content have a diminished capacity for contraction. Since the force of contraction is proportional to the breakdown of phosphocreatine, the poisoned muscles cannot develop and maintain normal tension. Since resynthesis of the hydrolyzed phosphocreatine is essential for the restoration of muscle excitability, the poisoned muscle is readily rendered inexcitable. Thus, as stated by Steiman, the low level of phosphocreatine and the marked interference with its resynthesis can readily explain the muscular weakness, fatigability, and paralysis resulting from exposure to lead. These observations lend new support to the view that the fundamental physiological lesion of lead palsy is in the muscle. It is interesting in this connection that the development of lead intoxication has been found to be accompanied by an increase in the urinary excretion of creatine. The impairment of phosphocreatine resynthesis in the muscle may be dependent upon a direct action of lead on intracellular enzyme systems."

Involvement of the central nervous system is evidenced by acute lead encephalitis. This pathology primarily attacks the meninges and produces edema of the brain and intracranial pressure. At post-mortem examination, Blackman* found vascular damage, exudation, and arterial changes similar to those seen in acute inflammatory processes. In cases of rapid lead absorption, capillary damage has been observed through retinal hemorrhage.

**Signs and Symptoms.**—In lead poisoning, the word "acute" or "chronic" cannot be applied with the same distinction that they connote in most other diseases. Only in rare instances does a workman suddenly develop this disease. On the contrary, the patient invariably notices that he is ill only because of the pain which is the result of intestinal colic. Yet it has been the experience of this writer that a careful analysis of the history of each patient will reveal that weakness, lassitude, and loss of appetite almost always precede colic. In other words, the onset is gradual, and certain disturbances have been present for a period of time before appearance of the acute symptom of colic.

Intestinal colic varies in its intensity and is usually intermittent in character. Waves of pain will be followed by a moderate sense of pressure, and then for a period of minutes, or even for an hour or two, the patient will be free of pain. Frequently, the patient has difficulty in localizing the pain and when asked to do so will rub his hand vaguely over the abdomen. In most instances the pain seems to centralize around the umbilicus. Abdominal tenderness is not marked and rarely is the abdomen distended. In two patients seen by this author it was possible to palpate the distended colon.

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*Blackman, S. S., Jr.: Intranuclear Inclusion Bodies in Kidney and Liver Caused by Lead Poisoning, Bull. Johns Hopkins Hosp. 58: 384, 1936.

Constipation is present in the majority of cases, although occasionally diarrhea is complained of. Nausea is fairly frequent, while vomiting is not common. Some writers mention bile in the vomitus, but this has never been observed in any of the author's patients. In the initial stages of intoxication, headache is almost always noted. The incidence of these and other signs and symptoms compiled from 164 of the author's cases are found in Table XI.

TABLE XI. INCIDENCE OF SYMPTOMS IN ONE HUNDRED SIXTY-FOUR CASES OF LEAD INTOXICATION

| SYMPTOM | PRESENT | ABSENT | SYMPTOM | PRESENT | ABSENT |
|---|---|---|---|---|---|
| Weakness | 131 | 33 | Stupor | 1 | 163 |
| Lassitude | 118 | 46 | Loss of memory | 1 | 163 |
| Anorexia | 130 | 34 | Neuritis | 7 | 157 |
| Colic | 123 | 41 | Lead line of the gums | 86 | 78 |
| Constipation | 102 | 62 | Pallor | 132 | 32 |
| Diarrhea | 9 | 155 | Jaundice | 0 | 164 |
| Loss of weight | 88 | 76 | Malnutrition | 61 | 103 |
| Insomnia | 15 | 149 | Abdominal tenderness | 58 | 106 |
| Headache | 142 | 22 | Blood in stool | 0 | 164 |
| Vomiting | 11 | 153 | Temporary hypertension | 36 | 128 |
| Nausea | 77 | 87 | Tremor | 20 | 144 |
| Vertigo | 28 | 136 | Wrist drop | 2 | 162 |
| Myalgia | 7 | 157 | Ankle drop | 0 | 164 |
| Nocturia | 5 | 159 | Joint tenderness | 4 | 160 |
| Impotence | 0 | 164 | Confusion | 1 | 163 |
| Numbness of extremities | 21 | 143 | Hallucinations | 1 | 163 |
| Visual disturbance | 1 | 163 | Coma | 0 | 164 |
| Nervousness | 120 | 44 | Ataxia | 1 | 163 |
| Convulsions | 1 | 163 | | | |

In Table XI the incidence of stupor, confusion, hallucination, visual disturbance, and ataxia is due to a single case of lead encephalitis. Myalgia and joint tenderness existing in the cases listed ceased after the acute phase of the illness in every patient except one, who maintained that he had pain in the extremities and especially in the big toe (right) for months after the exposure had ended. It was, however, doubted that this pain was due to lead poisoning. Thirty-six patients experienced an elevation of the systolic blood pressure above the normal, if their age is taken into consideration. Of this group, thirty-one were examined again six months after the onset of illness, and in each one the pressure had returned to a normal level. Five cases could not be followed.

1. THE BLOOD COUNT.—As previously stated, there usually occurs a reduction in the red blood cells, as well as a reduction of the hemoglobin, and, furthermore, a change in size and shape of the red blood cells. In lead poisoning, this alteration of the size and shape of the red cells is out of proportion to the reduction of the red cell or hemoglobin count.

The estimation of the stippled cells in a blood smear has become a routine procedure among physicians desiring to make a diagnosis of lead poisoning. In the normal stained blood smear, about 1 per cent of the erythrocytes show a bluish-pink color because of an affinity for the basic stain. This is the result of the presence of small amounts of the primordial, cytoplasmic basophilic substance persisting after the nuclei have been lost. An increase of these cells above the normal signifies a condition called polychromatophilia or polychromasia. This, as was pointed out, is a compensatory reaction, the yielding to a demand for a more rapid production of red cells with the result that erythrocytes are poured into the blood stream before they have reached maturity. We repeat the warning that these basophilic stippled cells *are not pathognomonic of lead poisoning,* for they are found as well in many other toxic states, in various types of anemia, and as a reaction to high altitudes.

McCord believes that the routine stippled cell determination could be improved upon by a method which is known as the "basophilic aggregation test." For those interested in the technique of preparing the smears for the basophilic aggregation test, the reader is referred to McCord's[12] original paper or to Johnstone's[13] textbook on *Occupational Diseases,* where a detailed description will be found. Regarding the utility of the basophilic aggregation test (often referred to as the B.A. test), McCord and co-workers[12] stated:

> "Polychromasia (polychromatophilia), punctate stippling, and reticulation are but different manifestations of one phenomenon—the presence of basophilic substance. The exact form of this basophilic substance existing in the unaltered blood is little known. Probably the picture observed as polychromasia after staining is nearest to the natural state of this material. The impression furnished by all available evidence is that of thousands of ultramicroscopic particles in acid suspension, or possibly in their innate form in acid solution. Reticulated cells probably are produced only as a result of laboratory manipulation and thus while not being true artifacts are laboratory creations. With suitable laboratory facilities one is able to observe through the microscope the actual formation of reticular processes in cells not previously exhibiting such reticulation. Through variations in laboratory technic, the experimenter may produce at will in a single blood smear all the well known varieties of reticulation, as fragmented, anastomosing, wreaths, mossy forms, etc. If the artificiality of reticulation be accepted as fact, then such terms as 'reticulocytosis' conceivably may be regarded as anomalous, in so far as these terms betoken the existence of reticular forms

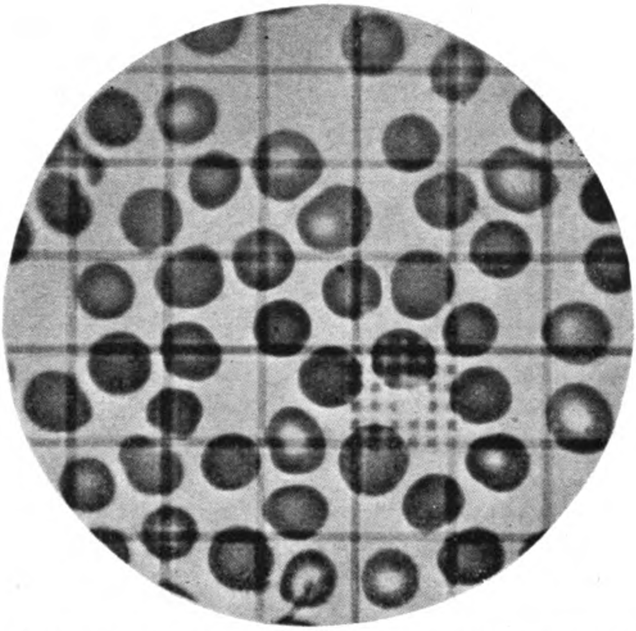

**Fig. 30.**—Section of fixed unlaked portion of blood smear used for the determination of total erythrocyte count and cellular abnormality. Photomicrograph with ×10 ocular and oil immersion objective. Straight lines shown are from Whipple grid used as a guide for quantitation. (Hyler and Bradley.)

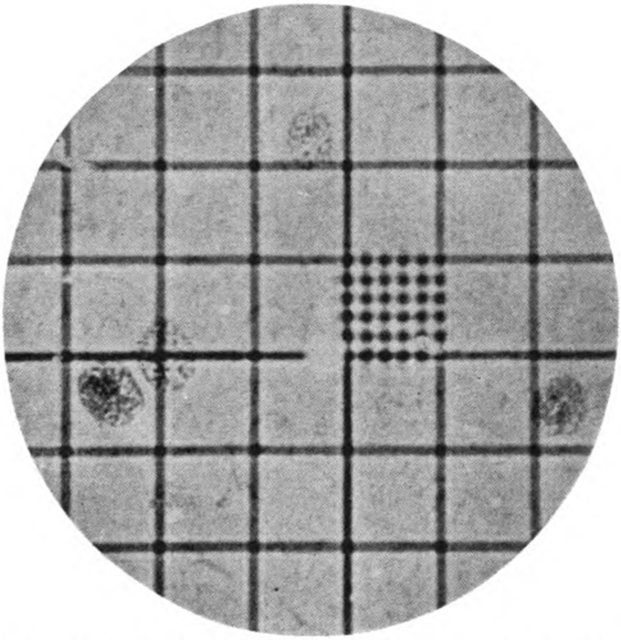

**Fig. 31.**—Laked portion of blood smear from the same slide as Fig. 30, showing four typical basophilic aggregations and faint outlines of nonbasophilic containing erythrocytes. (Hyler and Bradley.)

in the circulating blood. However, stippled cells are believed to exist as such in the unaltered blood. Through unknown processes, the ultramicroscopic particles observed as polychromasia or the basophilic materials in acid solutions, are caused to arrange themselves into masses characteristic of stippling.

"During 1934, basophilic aggregation tests were carried out simultaneously with stipple cell determinations, and in some instances with hemoglobin measurements and total red cell counts. Very early it was established that no consistent correlation was present. In many persons wholly unexposed to lead an occasional stipple cell was encountered and among lead workers approximately 90 per cent presented large numbers of stipple cells in the absence of clinical lead poisoning, and often after three months of no exposure to lead. The number of stipple cells in lead-using workers without clinical lead poisoning ordinarily was lower than 3,000 per million. The qualitative demonstration of stipple cells proved of no value in the diagnosis of clinical lead poisoning or in the making of decisions as to acceptability of men for continued work. Many workers regularly exhibiting stipple cells in their blood smears passed through the entire automobile production season without illness and without any lost time. In similar fashion it was found that no significant diagnostic values resided in the routine determination of hemoglobin percentages or in total red cell counts. Over and over high percentages of basophilic aggregations were detected with no significant changes either in the hemoglobin or in the erythrocyte count. For this reason all blood work other than the basophilic aggregation tests was abandoned as routine procedure in plant surveys, although complete blood work was carried out in the management of clinical cases of lead poisoning.

"The basic principle in the basophilic aggregation test is the enumeration of red blood cells containing basophilic substance, in contrast to the customary procedure of qualitative or quantitative examination for stipple or polychromatophilic cells [Figs. 30 and 31].

"The native state of basophilic material in unaltered red blood cells is not known, but in the process of laking and staining red cells this substance may be artificially aggregated into readily visible masses. In normal human adults, these aggregates rarely exceed 1 per cent of the total number of erythrocytes, but in lead exposed individuals the percentages ordinarily lie above this normal maximum, when considerable lead is being absorbed or when clinical lead poisoning is imminent. Findings of percentages above 1 to 1.5 per cent and especially above 2 per cent in persons exposed to lead at once suggest lead absorption and the possibility of approaching lead poisoning, or the actuality of early lead poisoning."

A determination of the reticulocytes rather than the stippled cells is recommended by some authorities who feel that a reticulocytosis is an earlier sign of plumbism than the appearance of punctate basophilia. The author believes that sufficient and conclusive information may be gained by utilizing the stippled count or basophilic aggregation test together with the determination of lead in urine or blood and, therefore, he has found it unnecesary to resort to a reticulocyte count. For the physician with a well-equipped laboratory and an efficient technician, the following technique for the determination of reticulocytes and stippled cells is suggested by the Division of Industrial Hygiene of the Illinois State Department of Health.

"Obtain a drop of blood from the lobe of the ear or the tip of the finger, and after the first drop has been wiped away, the second exuding drop is touched to the end of a clean slide, to be used as a spreader. Holding this slide at an angle of 30 degrees, bring the drop in contact with a horizontal slide near the right edge, and as soon as the blood runs along the line of contact, advance the spreader slide with an even sweep to the other end. The blood is drawn behind the edge of the advancing slide. An angle less than 30 degrees makes a thicker film; an angle more than 30 degrees makes a thinner film.

"After the smear has been *air dried,* it is ready for partial fixation. Fix half of the smear horizontally with absolute methyl alcohol (acetone free) as illustrated below. A thin line drawn through the middle of the dried film, from end to end, red or blue wax pencil, will prevent any possibility of the methyl alcohol passing up into the portion of the slide which is not to be fixed.

"In this fixed portion, the stippled cell count and red cell count will be made, while in the unfixed portion the reticulocyte count will be determined.

"The blood smear is now ready for staining, and for obtaining accurate counts, it should be stained as soon as possible after air drying.

"The formula for the stain to be used is as follows:

| | |
|---|---|
| Methylene blue chloride | 0.50 Gm. |
| Borax | 0.05 Gm. |
| Loeffler's methylene blue[*] | 5.00 cc. |
| Distilled water | Full 100 cc. |

"When filtered it is ready for use. Should keep many months. Does not deteriorate very readily.

"The smear is immersed in the stain for twenty minutes, washed well in distilled water, and dried either by air or with blotting paper. Overstaining is not possible; however, twenty minutes are sufficient for maximum results. The blood smear is now ready to be examined, but before this is attempted it becomes necessary to calibrate the microscope you use. An Ehrlich eyepiece or a set of Ehrlich stops should be used for counting. The Ehrlich ocular is more convenient and is recommended for this work. The eyepiece is constructed with different sized squared openings marked 1, 2, 3, and 4. The size of the opening is changed merely by turning the adjustment. Number 1, the small opening, is used for making the red cell count, and Number 3, the large opening, is used for the reticulocyte and stippled cell counts.

"It is also necessary to use a graduated mechanical stage. The calibration is very simple for it is only necessary to determine the number of fields over a distance of 10 mm. by moving the graduated mechanical stage in a horizontal plane. That is, for example, in moving from the 20 mm. mark to the 30 mm. mark, 95 fields are counted. These 95 fields represent the total number of fields counted every time we make an examination through any 10 millimeters. By utilizing this type of mechanical stage, it is easy to standardize procedure by computing the number of fields over a distance of 10 mm. on the graduated mechanical stage by moving it horizontally.

"Next, adjust the Ehrlich eyepiece to the Number 3 opening for it is with this opening you will make your calibration. Take the stained slide, focus it under the oil immersion lens, and set the mechanical stage, say at 30 millimeters. Notice the cells along margin A, then move position of slide, placing the cells noticed at margin A over to margin B. This constitutes field 1. The same procedure is continued until 10 mm. have been covered. The number of fields counted in this

---

[*] Formula for Loeffler's methylene blue:

| | |
|---|---|
| Saturated alcoholic solution methylene blue | 30 cc. |
| 1:10,000 solution KOH | 100 cc. |

(Two drops of a 10 per cent solution of KOH in 100 cc. of water makes a 1:10,000 solution.)

10 mm. must be recorded and you will then have the permanent calibration for this particular microscope. The same procedure will have to be followed to calibrate any other new or different microscope to be used for this purpose.

"After the calibration has been completed, the slide is ready for counting. You already have a stained smear, one-half of it fixed, the other half unfixed, and the Ehrlich eyepiece set at the Number 3 opening. First locate the unfixed portion, for the reticulocytes should be counted first; then set the mechanical stage say at the 30 mm. mark. Move in a horizontal plane, counting all the reticulocytes as you proceed to the 40 mm. mark. Now, for example, if in going through this operation from the 30 to the 40 mm. mark you have counted 66 reticulocytes, this represents the total number of reticulocytes in the 10 mm. or in 95 fields (using the ocular Number 3 opening). Next, adjust the opening on the eyepiece to the Number 1 stop and move the slide to the fixed portion of the smear, remaining in the same 10 mm. in which you made the reticulocyte count. You are now ready to make the red cell count. It is convenient, therefore, to start at the 40 mm. mark and work back to the 30 mm. mark, for the 40 mm. mark was the point where the reticulocyte count ended. It is only necessary here to count at random any 20 fields while you move the slide in a horizontal plane from the 40 to the 30 mm. mark. For example, if the total number of red cells counted for any 20 fields totaled 300, the average number of red cells per field would be 15 (with the Number 1 ocular opening).

"The stippled cells are counted in the same manner as the reticulocytes were counted, except that they are counted in the fixed portion of the smear. The Number 3 ocular opening is also used in this determination.

"After the reticulocytes, stippled cells, and red cells have been counted, the calculation proceeds as follows:

"The reticulocytes are reported in per cent and the stippled cells are reported as so many per 100,000 red cells. We already said that if we counted 300 red cells in 20 fields, the average would be 15. We then multiply 15 by 9, giving us 135 red cells, because ocular opening Number 3 is 9 times greater than ocular Number 1. We now have the average number of red cells corresponding to the same sized field in which is made the reticulocyte count. However, the total number of reticulocytes represented not 1 but 95 fields. It is, therefore, necessary to multiply 135 by 95 to give us our total red count for 95 fields, which is 12,825. Furthermore, we found 66 reticulocytes and 12,825 red cells in the fields. By dividing the number of reticulocytes by the number of red cells and multiplying by 100, the following percentage is obtained:

$$\frac{66}{12{,}825} \times 100 = .51\% \text{ reticulocytes}$$

"The stippled cell count is calculated in the same manner. For example, if we found 12 stippled cells, we divide this total by the total number of red cells.

The result is $\frac{12}{12{,}825} = 0.00093$. This is equivalent to 93 stippled cells per 100,000 red cells.

"Normal reticulocyte count is 0.1 to 0.9 per cent. Above 0.9 per cent may indicate lead is being absorbed. Counts running above should be checked for increase, for anything above begins to show absorption, the per cent becoming greater as absorption increases.

"Normal stippled cell count is 1 to 15 per 100,000 red cells. The greater the number of stippled cells, the greater the absorption."

2. Lead Content in the Urine.—If carried out carefully, the analysis of urine for its lead content constitutes the most reliable index of the degree of acute lead intoxication. It is considered a more valuable test than that of the estimation of lead in whole blood or in serum, because the change in the rate of urinary lead excretion in response to lead absorption is greater proportionately than the change in lead concentration of the blood.

One cannot be too careful in collecting a twenty-four hour specimen of urine for lead determinations. It should be collected in a large Pyrex container, and all dust or contamination of any form should be avoided. If it is necessary to resort to rubber or metal catheters, these should be free of lead in their composition. Several consecutive analysis of a twenty-four hour specimen should be made. It is also advisable to check the results against whole blood determinations. A modified dithizone titrimetric extraction is the method usually employed in urinary lead analysis.

The range of normal values of lead excretion varies considerably in the opinion of the various investigators. For many years the upper limit of normal was considered to be 0.05 mg. per liter for samples of one liter or more. In fact, even today most clinical laboratories have not changed these figures on their printed forms. The Committee on Lead Poisoning* appointed by the American Public Health Association, however, stated that:

> "In the upper limit of the normal lead concentration the urine approximates 0.08 mg. per liter for samples of one liter or more, and 0.12 mg. per liter for spot samples of about 100 ml. in volume (allowing for some degree of concentration of the sample). Lead in concentrations below 0.15 mg. per liter has doubtful significance, especially if the lead exposure had continued up to or nearly to the date on which the sample of urine was obtained. Hazardous lead exposure usually induces lead excretion at levels above 0.15 mg. per liter, and the large proportion of cases of lead poisoning examined during an episode of intoxication show concentrations between 0.15 and 0.30 mg. per liter. Concentrations in excess of 0.50 mg. of lead per liter of urine are encountered infrequently, and always require verification by additional analyses."

A rapid microanalytical method for lead in urine has been devised by Kaye.[14] The method is based on the total precipitation of lead by the addition of aqueous ammonia to urine. Lead phosphate is easily filtered out with the calcium and magnesium phosphates. This precipitate is dissolved with hot dilute nitric acid and is heated to oxidize organic material. Citric acid is added to keep the phosphates in solution during later neutralization, and potassium cyanide is added to form complexions with interfering metals. When the pH is adjusted at 9-10, lead is extracted with dithizone to separate it from extraneous salts and interfering substances. Lead is then split from the dithizone and titrated with standardized dithizone. Under the above conditions only bismuth, tin, thallium, and lead react with dithizone. To remove the interference of bismuth and tin, these are previously extracted with dithizone while the solution is still acidic. Lead is not extracted in this acid solution. Thallium, as a source of interference, has not been removed but its occurrence is unlikely.

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* Occupational Lead Exposure and Lead Poisoning, New York, 1943, American Public Health Association, p. 15.

3. Spot or Instantaneous Urine Analysis.—In large plants or where a great number of men are to be constantly observed, it is difficult to obtain twenty-four hour specimens without having them become contaminated or having inaccurate amounts collected. Realization of this difficulty with the workmen at the Westinghouse Electric and Manufacturing Company, at East Pittsburgh, led Barnes[15] to investigate the possibility of correlation between short-time or instantaneous specimen and average twenty-four hour excretions. It was felt that some correlation might be obtained if factors such as time of day, acidity, specific gravity, or total nitrogen were taken into account. After experimenting with exposed, as well as unexposed persons, Barnes[15] concluded that a determination of the lead excreta per hour, as found by analyzing a specimen representing two or three hours' excretion during the working day, gives a fairly reliable measure of the average excretion for that day. It impresses the author that this procedure suggested by Barnes can serve the plant physician where all the factors can be controlled, but that for a single examination of a patient suspected of lead poisoning the twenty-four hour specimen should be obtained.

The *instructions* that are to be adhered to in obtaining these spot or instantaneous specimens are as follows:

1. Have the patient empty the bladder. This first specimen is obtained for the purpose of starting at a definite time with an empty bladder and is not to be used in the lead analysis. The first specimen should not be placed in the special container. It may be used for the determination of sugar, albumin, specific gravity, etc., if desired. Record on a data sheet the time that the employee emptied the bladder. The date, the employer's name, and the occupation should also be recorded.

2. Instruct the employee to return to the medical department at least two hours later (preferably three to four hours later). He should not void the urine until he has returned to the medical department, where the specimen will be taken.

3. When the employee returns, mark the time on the data sheet and have him void directly into the special container. Special care should be exercised to be certain that no dust or dirt is dropped into the container. These containers have been specially cleaned at the laboratories and should not be rinsed or cleaned in any way. Do not add any preservative. Do not remove any of this sample from the special container, and do not use any of it for other tests.

4. The results of the tests are reported in micrograms of lead excreted per hour.

4. Blood Lead Examination.—Here, again, in the technique of collecting the blood sample, contamination should be avoided and the use of lead-free containers, syringes, and needles is required. Reliance may be placed only in a laboratory or chemist qualified to perform this test, for not all clinical laboratories or hospitals are equipped or manned to carry out this type of analysis.

Blood lead concentrations in the individual are subject to but little variation from hour to hour, except in response to brief, intense lead exposure, and therefore samples may be obtained for analysis at any time of day. The normal lead concentrations in the blood, as previously indicated, range from 0.01 to 0.06 mg. per 100 Gm. of whole blood, with only occasional values in excess of 0.04 mg. Results in excess of 0.07 mg. per 100 Gm. of whole blood are indicative of lead exposure within some recent period, while results in excess of 0.10 mg. per 100 Gm. show that the exposure has been considerable. Results approximating 0.50 mg. per 100 Gm. are unusual but may occur when the industrial (or other) exposure is quite severe. Results much above this level are open to question as to their accuracy, and except in very rare instances of overwhelming accidental exposure, results in excess of 1.00 mg. per 100 Gm. must be regarded as evidence of contamination of the sample in the process of collection or analysis. Doubtful results should be checked by the analysis of additional samples, or by analysis of the urine, or by both means.

5. Lead Analysis of the Air.—The most common appliances of collecting dusts for lead determination are the Greenburg-Smith impinger, the midget impinger, and the electrostatic precipitator. Several samples should be made of each area of exposure. The upper limit of permissible lead concentration in the air is 1.5 mg. of lead per 10 cubic meters of air.

**The Basis for Diagnosis.**—There are more cases of alleged lead poisoning accepted as actual cases of poisoning because of the failure of physicians to distinguish between *lead absorption* and *lead intoxication.* Since lead is a natural constituent of the soil, water, vegetation, and air, some degree of lead exists normally in human tissues. Individuals under study should, therefore, be classified under three headings:

1. Normal lead absorption
2. Abnormal lead absorption without evidence of intoxication
3. Abnormal lead absorption with evidence of intoxication

Intestinal colic, a lead line of the gums, and basophilic stippling have too long been accepted as evidence of lead poisoning. It must be repeated again that these are not "proof-positive evidence of lead intoxication."

In an exposed individual they constitute suggestive evidence only. When lead absorption has been sufficiently heavy, deposits of sulfide will occur along the gingival margins in individuals with defective oral hygiene. But this is a sign of absorption only. In low levels of absorption the line is likely to be indistinct but will become more pronounced and easily discernible as the absorption increases. A hand lens should always be used in examining the gums for this "blue line."

Although intestinal colic is a characteristic symptom of lead poisoning, its occurrence in a lead worker should not be blindly accepted as indicating lead intoxication. Proper treatment of appendicitis or other abdominal pathology has often been delayed because colic was automatically considered to result from lead intoxication.

The various possible causes of basophilic stippling have been discussed previously. While this procedure is important in the estimation of the degree of absorption in acute cases, it must not be considered a conclusive test. However, in the hands of experienced plant physicians, it is of invaluable aid in the routine check of their workers for increased absorption.

In an accurate diagnosis, the history must be carefully elicited and frequently checked by environmental studies. Workmen are not always cognizant of the degree or type of their exposure. Men who may be employed in an occupation which does not require them to handle lead may be exposed to the fumes or dust created by another worker. Or the exposure may not be occupational, as a case of ours illustrates. There was no question in this case that the patient had lead intoxication, but environmental studies failed to disclose a source in his occupation. After a considerable amount of investigation it was found that he has set up a welding outfit in his home garage where he had been welding on metal painted with red litharge.

Laboratory studies should include a blood count to determine the presence or absence of anemia and its possible degree. Either urine or whole blood or both should be tested for their lead content. Erroneous conclusions can be avoided if both substances are tested. For medicolegal purposes it is advisable to hospitalize the patient in order to guarantee adherence to a desired technique.

**Differential Diagnosis.**—The one symptom which is most likely to be misleading is that of pain in the abdomen. Lead colic must be differentiated from renal colic, appendicitis, gastric or duodenal ulcer, acute gastroenteritis, etc. In one case in our series, for instance, pain due to coronary occlusion was initially attributed to lead colic.

Peripheral neuritis in one exposed to lead must be differentiated from neuritis due to infection, toxemias other than from lead, malnutrition, and disturbed metabolic states. Peripheral neuritis due to lead absorption is usually painless and most often confined to the extensor muscle groups of the forearms.

Lead encephalitis is rare and is the result of severe exposure. In lead encephalitis, the lead content in the urine and blood should be higher than is found in the ordinary case of lead intoxication. Kehoe[16] believes that the analysis of the cerebrospinal fluid is of doubtful diagnostic value because of the difficulty in obtaining adequate and uncontaminated samples, and because of the somewhat uncertain significance of the results in relation to absorption. Encephalitis due to such other causes as brain tumor, infection, syphilis, uremia, and other intoxications or poisons must be ruled out.

**Sequelae of Lead Poisoning.**—In the field of medicine, one function of a new textbook is to replace old, unsubstantiated concepts with the most recent authoritative opinion or experience. Lead poisoning is a profusely documented subject and the passing years have permitted the inclusion of many contentions now recognized as unfounded. To reprint these older, but baseless concepts, as being worthy of equal consideration with recent, adequately studied opinions only leads to confusion and false impressions. An example of such unnecessary reference is found in a recent monograph on lead poisoning where it is stated: "Workers in lead industries appear to have a relatively high incidence of diseases of the respiratory tract, particularly chronic bronchitis, emphysema, asthma and, according to some, bronchopneumonia." The authors of the foregoing statement do not cite this passage as being their own experience, but attribute it to a German and a Swiss physician. Yet such a contention is not within the American experience. For some years the effect on the respiratory system of the various industrial dusts and chemicals has been studied by the Trudeau Foundation at Saranac, New York. At the 1942 Symposium, Belnap[17] stated that "lead absorption, even complicated by disabling lead intoxication, does not reduce general resistance to upper respiratory infection, or non-specific pulmonary infection. It does not predispose to or aggravate pulmonary tuberculosis."

It is not intended here to suggest that those pathologic or physiologic possibilities which as yet have not been scientifically exhausted should not be discussed. But why, for instance, should textbooks still suggest that lead causes arteriosclerosis when all recent data point to the contrary? It has been demonstrated that intestinal colic, wrist drop, and certain other effects may result from undue lead absorption. These and a number of other phenomena have been firmly established by time. Likewise, time and modern methods of conducting surveys together with the practice of using control groups for comparison have proved that we can disregard most of the old *impressions* of the sequelae of lead intoxication.

It is now well recognized that lead poisoning is primarily of three types, namely, the alimentary type, the neuromuscular type, and the rare encephalopathic type, as has been pointed out in this chapter. Common to all these types is the disturbance to the red blood cells. The author also believes that concomitant with the first two types and obviously with the third one goes a temporary toxic effect on the nervous system as evidenced by headache, insomnia, nervousness, and mental depression. This effect disappears in a very short time without residual stigmas.

In support of what has been written here in regard to sequelae, it can be stated that after many years devoted entirely to occupational medicine and a personal study of a large group of workers suffering from lead intoxication, this writer has never seen a single instance of sequelae of any type except for one case of permanent wrist drop. The reasons for this are efficient preventive measures which are now the rule in industry. Even in the absence of medical or engineering supervision, workers today seek early medical care if any suspicion of an occupational illness arises. The Labor Unions emphasize that this is one of their rights.

Before terminating this discussion on the sequelae of lead poisoning, it is necessary to present briefly certain recent observations dealing with a few of the formerly alleged sequelae.

ARTERIOSCLEROSIS AND HYPERTENSION.—It is not difficult to explain how the belief has come about that lead causes arteriosclerosis. Physicians practicing in the industrial centers of Europe, many years ago, observed that when the "old stagers" came to autopsy, a fairly high incidence of arteriosclerosis was observed, which they falsely attributed to lead rather than to the natural degenerative processes of that age group.

Modern opinion is not based upon such chance observations. It comes from the experience of men like Belnap[18] who have had extensive experience in the lead industries. Belnap compared a large group of lead workers with other workers of similar age and economic circumstances whose occupation did not bring them into contact with lead and he came to the conclusion that there exists no difference as to arteriosclerosis or hypertension. Dr. May Mayers,[19] Chief of the Medical Unit, Division of Industrial Hygiene of the New York State Department of Labor, and a member of the Committee on Lead Poisoning of the American Public Health Association, examined 381 workers who had been exposed to lead for a considerable period of time, and stated that while arteriosclerosis occurred earlier in all types of industrial workers as compared with office workers and professional men, there was no evidence that lead played any part in the development of arteriosclerosis or hypertension. In a more recent publication[20] the same author writes "that prolonged exposure to lead accelerates the development of arteriosclerosis is an old medical concept which has not been confirmed by recent investigation."

Dreessen[21] made a survey of six lead battery plants for the United States Department of Health. In the group surveyed, 12 per cent had been employed for twenty years or more and 75 per cent for five years or more. He found that neither arteriosclerosis nor hypertension was more prevalent in the exposed group than in a control group.

It should be explained that this discussion of hypertension pertains to the permanently or persistently elevated blood pressure levels, not to the temporary disturbance which is fairly common in the acute stage. Since such hypertension is noted in workers suffering from intoxication after only a short exposure, it can obviously not be due to arteriosclerosis. Some authors feel that this temporary rise in blood pressure is a reaction to pain, but it seems more likely that it is the result of toxemia.

NEPHRITIS.—While albuminuria and polyuria may, at times, occur in the acute stage, there exists no evidence that excessive lead absorption causes any chronic renal damage.

GASTRIC OR DUODENAL ULCER.—Lead does not produce ulcers in the intestinal tract. The collected data of various observers fail to report any instance of gastric or duodenal ulcers resulting from lead exposure.

CARDIOVASCULAR SYSTEM.—In the writer's experience, no evidence has been submitted to the fact that lead exposure produces permanent damage to the heart or to the vascular system.

MULTIPLE SCLEROSIS.—The occasional case of multiple sclerosis in a lead worker has given rise to speculation as to a possible relationship. According to Mayer, recent studies indicate that no such relationship exists.

**Treatment.**—Some years ago the author attempted to "store lead" in a subject, and then subsequently "delead" the patient. In recent years this procedure has been discarded due to the belief that lead is excreted from the body by normal physiologic processes, and that no type of therapy influences this action. The regime now followed is not concerned with the lead *per se* but rather with the incidental effects of lead. The regime is as follows:

1. Remove the patient from exposure.
2. Hospitalize the patient for purposes of study, or whenever the intoxication is severe.
3. For intestinal colic, calcium gluconate, 10 cc. of a 10 per cent solution intravenously, is given every four hours. Although the frequency of administration may be reduced with the subsiding of symptoms, medication with calcium is continued for at least two days after cessation of the colic.
4. For constipation, magnesium sulfate, ½ ounce daily, is administered.

5. For anemia, small doses of iron are given and continued until repeated blood counts reveal a return to normal of the red cells and hemoglobin.
6. The patient is returned to work, but not to a lead exposure, as soon as his clinical condition warrants this change.
7. At no time is morphine or atropine given for abdominal discomfort. For excessive nervousness, one of the barbiturates is used. For headaches, aspirin.

As previously intimated, no attempt is ever made to "delead" the patient. In our experience this is not necessary and its efficacy is questionable. Since, however, there are those who still indulge in this procedure, the various viewpoints are herewith presented.

DELEADING BY HIGH CALCIUM DIET.—Following the work of Aub, Fairhall, Minot, and Reznikoff,[4] which indicated that lead could be either stored or excreted by distorting the calcium metabolism, therapy was directed along these lines. The conclusions drawn from their work were that a negative calcium balance increases the rate of lead excretion, while a positive calcium balance favors the storage of lead. To this end, then, the treatment for the acute episode in the past has consisted of the following:

1. A high calcium diet, which includes much milk, green vegetables, and potatoes.
2. The administration of calcium products and vitamin D orally; for example, calcium lactate, 1 drachm (4 Gm.), once every two hours; or calcium gluconate, 7½ grains (0.5 Gm.), in milk at three-hour intervals, so that the patient obtains five or six doses daily.
3. Calcium gluconate, 10 cc. of 10 per cent solution, intravenously daily for five days.
4. Magnesium sulfate, ½ ounce every morning, to sweep out the relaxed bowel.
5. As substitutes for the intravenous calcium gluconate in the relieving of abdominal pain, hypodermic injections of morphine sulfate, ¼ grain (0.015 Gm.), and atropine sulfate, 1/100 grain (0.0006 Gm.); nitroglycerin, from one to two 1/100 grain (0.0006 Gm.) tablets dissolved under the tongue; or the inhalation of the contents of a 5 minim (0.32 cc.) amyl nitrite pearl.

*Disadvantages of Method.*—Recently much doubt has been thrown on this method of treatment by the work of Lederer and Bing.[22] Their work indicated that the deposition of lead in the bones of growing animals is retarded by increasing the calcium content of the diet and that the phosphorus content of the diet had no significant effect on the amount of lead deposited in the bones. This would indicate, then, that a high calcium diet would be the optimum diet for deleading, rather than for the deposition of lead in the bones. The use of the high calcium diet, then, according to this view, might actually be dangerous by mobilizing lead for possible storage in the central nervous system in the acutely ill patient. Taeger[23] arrived at such a conclusion and advised against large doses as well as against the intravenous administration of calcium. Shelling and Hopper[24] believed the use of calcium did not induce superior calcification, but rather had the opposite effect, especially when the phosphorus intake was inadequate. Kehoe and Thamann[25] were of the opinion that the use of agents to promote the quick release of lead from the tissues was hazardous. Shelling[26] demonstrated experimentally that the addition of calcium carbonate to an optimal stock diet containing lead carbonate fed to rats resulted in an increased toxicity. These rats evidenced toxicity and died sooner than the rats fed on the same stock diet to which sodium phosphate had been added. Gray and Greenfield[27] reported a small series of cases where prolonged administration of high calcium diet therapy was responsible for permanent, irreparable damage to the nervous system. They concluded that a high calcium regimen was the best means of deleading. In view of this evidence, the advisability of the use of a high calcium diet alone is open to question. The frequently dramatic relief of abdominal pain obtained from the intravenous administration of calcium may represent, then, only a general depressant action of calcium on smooth muscle rather than a specific reaction as regards the lead in the blood stream.

With High Phosphorus and High Vitamin.—Gray and Greenfield[27] found the use of an optimal calcium diet, with a high phosphorus and high vitamin diet (D particularly), the best treatment in these cases. With this regimen an essential element was the administration of sodium phosphate, 2 drachms three times daily. Shelling[26] indicated that in order to deposit lead in the skeleton as an insoluble lead phosphate, a certain amount of phosphate was required, and thus when phosphate was inadequate, free lead might remain in circulation. He stated that the addition of sodium phosphate "proved sufficient phosphate for the deposition of both calcium and lead phosphate in the skeleton, for the excretion of lead as the relatively non-toxic lead phosphate, and also for the formation of colloidal lead phosphate in the blood." As indicated, the work of Lederer and Bing[22] directly opposes this statement and seems to indicate that the addition of phosphate had no significant effect. The work of Lederer and Bing further indicated that the beneficial effect of calcium in the diet in retarding the retention of lead by the body was due to reactions which occurred in the intestinal tract and that calcium carbonate orally was the most valuable preparation.

Deleading by Low Calcium Diet.—In the past a regimen of a low calcium, high phosphorus diet, phosphoric acid, and magnesium sulfate had been used successfully by Gray[28] in deleading treatment. He used the diet shown in Table XII with claims of success.

He also elaborated a similar diet with a calcium or phosphorus ratio of 1:4. To these were added 20 minims of viosterol (250 D) daily, since vitamin D was deficient in the diet. Previously he had used a low calcium diet consisting of meat, liver, potato, rice, tomatoes (cooked without milk), canned corn, bananas, apples (peeled), tea, coffee (without milk), butterfat, bread (prepared without milk, such as salt-free nephritic bread or sodium bicarbonate biscuits or crackers), sugar, salt, and pepper. Phosphoric acid in amounts of from 10 cc. every two hours for six doses daily to 10 cc. once every hour for ten doses daily was used. In addition, magnesium sulfate, ½ ounce (15 Gm.), each morning was given. With this regimen, increase in urinary lead was at first seen, and this was followed by a decrease as the patient was presumably deleaded.

TABLE XII

LOW CALCIUM-HIGH PHOSPHORUS DIET (RATIO 1:3)

| FOOD | WEIGHT (GM.) | FOOD | WEIGHT (GM.) |
|---|---|---|---|
| Applesauce | 100 | Lamb chops | 160 |
| Grapes | 103 | Liver | 90 |
| Banana | 100 | Bacon | 15 |
| Pineapple juice | 540 | Baked potato | 100 |
| Shredded wheat (1) | 27 | Tomatoes | 100 |
| Whole-wheat bread | 120 | Green peas | 100 |
| Macaroni | 30 | Mushrooms | 60 |
| Egg yolks (4) | 80 | Celery | 30 |
| Butter | 30 | Almonds | 15 |
| Cream, 40 per cent | 70 | Oranges | 200 |
| Lettuce | 30 | | |

Total: Protein, 101.78 Gm.; calcium, 0.601 Gm.; phosphorus, 1.889 Gm., calories, 2,969.88.

WITH OTHER SUBSTANCES.—Other methods suggested for deleading have included the low calcium diet cited; ammonium chloride, 15 grains (1 Gm.) every hour for ten doses daily; and magnesium sulfate, ½ ounce (15 Gm.), every morning. Belknap[29] used fifteen drops of potassium iodide twice daily or seven drops twice daily of sodium iodide in saturated solution. Sodium bicarbonate, from 5 to 8 drachms (20 to 30 Gm.), daily, divided into five or six portions, has also been used as a parathyroid extract.

*Advisability of Deleading.*—As indicated in the foregoing, there is disagreement concerning the possibility of deposition of lead in bones by changes in diet or by medication. In addition, the advisability, as well as the possibility and the methods of deleading, is distinctly open to question. Most authorities feel that it should not be attempted before from three to four weeks have elapsed after the acute episode. Most feel that the deleading should be carried out only in the hospital and with adequate laboratory checks, although Belknap[29], deleads patients in mild cases while they are ambulatory but under close supervision. He requires a blood level of 80 per cent hemoglobin, 4,000,000 or more red blood cells, stippled cells not more than from 1,000 to 5,000 per million red blood cells for two to three weeks, and a twenty-four hour urine lead 0.15 mg. or lower before undertaking the deleading. He also deleads no oftener than once in four weeks, and preferably not oftener than once in six or eight weeks.

*Benefits Versus Drawbacks of Deleading.*—Those who oppose deleading feel that the danger of producing lead encephalopathy is too great, and that the patient cannot be successfully "deleaded" at any rate. Those who favor it urge that the stored lead represents a constant potential hazard, since acute episodes, such as infections with acidosis or other periods of metabolic stress, may cause deleading at an undesirable time. Aub[30] feels that thorough deleading approximately halves the period of disability for lead palsies.

ASCORBIC ACID THERAPY.—Holmes, Campbell, and Amberg[31] found 100 mg. of ascorbic acid daily of much benefit in lead intoxication cases, and also observed that the ingestion of alcohol had an unfavorable influence and in several instances nullified the benefits of treatment. Others have had no success with this form of therapy.

**Treatment in Lead Palsy.**—In this condition, stretching of fibers of paretic muscles should be prevented. In those cases presenting a wrist drop, a cocked-up splint, to include the fingers and worn night and day, is used until function returns. Hot-water baths (110° F.), electric stimulation by interrupted galvanic current, and gentle massage, preferably by whirlpool, once every day or two are used. Strenuous massage and violent exercise are avoided. Re-education exercises are of value.

**Treatment in Lead Encephalopathy.**—Encephalopathy is rare, and has been seen only once in our clinic. When it does occur, lumbar puncture and sedation may be required. Magnesium sulfate, 2 cc. of a 25 per cent solution given intramuscularly from every four to six hours, and barbiturates are used. For convulsions, barbiturates, for example, sodium amytal, from 3¾ to 7½ grains (0.23 to 0.5 Gm.), are given intravenously, and avertin is used rectally. Suboccipital or subtemporal decompression in lead encephalopathy has been used successfully.[32]

**Prophylaxis.**—The best method to avoid lead poisoning is, of course, to take measures to prevent it. To this end, Gant[33] suggests the following rules and mechanical features:

"1. Hoods with adequate exhaust ventilation to be at any point where dusts or fumes arise;

"2. All mixing and shaking to be done in enclosed machines;

"3. All powdered compounds to be transferred by means of adequate vacuum lines;

"4. Work tables to be provided with grated tops, ventilated from below, and equipped with small narrow troughs filled with water, and attached to each side to prevent dust falling to the floor;

"5. All inlets to exhaust ventilating ducts to be below the level of the nose, as far as possible, to prevent fine dust particles from being inhaled;

"6. A chemical analysis of samples of air from various parts of the plant to be made at different intervals during working hours (any spot showing more than 1.5 mg. of lead per 10 cu. m. is a potential health hazard);

"7. Employees in hazardous spots, as found under '6' to wear respirators. Where movements are confined to a small area, forced-draft respirators (fresh air from the outside) to be used. If exposure is to fumes, masks to be provided with canisters charged with activated carbon. (Respirators are only from 70 to 90 per cent efficient and must be inspected and changed frequently);

"8. No dust allowed to accumulate on the floor;

"9. Floors to be wet at frequent intervals, and powdered compounds used, if possible;

"10. All cleaning to be done with large vacuum lines. No sweeping;

"11. To prevent the collection of dust on rafters, sills, etc., ventilating ducts to be installed, equipped with dust-filters near the ceiling or in mid-air;

"12. To avoid the contamination of outside air and surrounding territory, and as an economy measure, electrostatic precipitators to be installed in the stacks and flues;

"13. The personal hygiene of the employees to be closely guarded. Locker-rooms with showers, a lunchroom isolated from the plant, and a change of overalls twice a day to be provided. Hands and face to be washed before eating. No eating, chewing, or smoking during working hours."

### Case Histories.—

CASE 1.—S. H., a 27-year-old Negro man, was first examined on Jan. 8, 1945, at which time he complained of severe abdominal cramps, headache, and insomnia. Two weeks prior to the examination he had noted that he had no appetite and was having difficulty in sleeping. A few days later he developed headaches, and on two occasions he vomited. He had suffered from constipation for several years. One week prior to examination he first noticed a dull abdominal pain which increased daily in severity. He stopped work on Jan. 4, 1945.

This patient had worked for three months for a lead recovery plant where he operated a sack-loading machine which filled sacks with lead dust. He claimed that he used his respirator faithfully, but he believed it "weren't no good no more."

The physical examination of this workman was entirely negative except for a blood pressure of 170/80 and mild tenderness in the area of the umbilicus.

Laboratory studies revealed: hemoglobin, 70 per cent; R.B.C., 4,100,000; basophilic aggregation, 2.5 per cent; lead in the twenty-four hour urine, 0.21 mg. per liter; and the lead in the whole blood, 0.178 mg. per 100 cubic centimeters.

*Comment.*—The patient was hospitalized and received 10 cc. of 10 per cent calcium gluconate intravenously, q.i.d. At the end of two days he requested that he be allowed to go home, because he "felt good." He was permitted to do so but was ordered to return daily for three more days for intravenous administration of calcium gluconate. He was likewise given iron therapy orally.

Fifteen days after his release from the hospital a re-examination showed the blood pressure to be 124/78 and the patient free from symptoms. He returned to work, although not to a lead exposure. It will be noted that the patient received no treatment with exception of that for anemia and colic.

CASE 2.—Peripheral neuritis and wrist drop: This case has been reported extensively in Part One, Chapter VI, The Basic Approach to a Diagnosis of Occupational Disease.

CASE 3.—O. B., a 49-year-old man, was admitted to the author's service in a semicomatose condition. According to his wife, he had evidenced a changed personality for the last three weeks prior to admission. Whereas he had once been alert, attentive, and an avid reader of the newspaper, he had now become slovenly, indifferent and apathetic, and would come home from work to sit around in a drowsy condition. Two weeks prior to admission he had developed severe headache and frequent vomiting.

The initial examination revealed an extremely pale, undernourished male weighing 127 pounds (normal weight, 155 pounds). The pupils reacted to light in a sluggish, delayed manner. The fundi showed papilledema, more marked on the right. The optic vessels were dilated and the outline of the discs obscured. Visual field tests proved unreliable.

The blood pressure was 160/90; the heart rate 88, with a hemic murmur in the pulmonic area. The abdomen was soft and no organs or masses were palpated. During a lucid moment the patient complained of generalized abdominal tenderness.

The initial neurologic examination revealed an inarticulate, disoriented, and restless patient who, during the first twenty-four hours of hospitalization, had three fairly severe seizures of colonic spasm. When not in an actual seizure he had small, jerking movements of the arms and legs. No rigidity of the neck was noted. The temperature was 99° F., and it never rose above that. Testing of the various cranial nerves proved unsatisfactory. All superficial and deep reflexes were present and equal, but the knee jerks were hyperactive. Subsequently, when the patient had recovered to a point where he could stand, the Romberg test was negative. At first, however, the patient could not walk; his gait was staggering and he would fall after a few steps.

Following the first spinal puncture he became lucid enough to state that the headache had been relieved. However, he remained disoriented, and verbal instructions were poorly perceived. He could not write his name or recognize written words and was unable to copy a simple drawing or draw a circle or a square. During lucid moments he claimed that he could not see well—that things seemed hazy and foggy.

Following the second spinal puncture, the mental condition cleared rapidly, the headaches disappeared, and the patient became cooperative. The spasms did not return.

An x-ray of the chest was essentially negative.

Blood count: hemoglobin, 55 per cent; erythrocytes, 3,800,000; leucocytes, 7,200; polynuclear neutrophiles, 66 per cent; lymphocytes, 31 per cent; large mononuclears, 2 per cent; eosinophiles, 1 per cent; marked variation in size and shape of the red blood cells.

Urine: basophilic aggregation, 3.8 per cent; lead in whole blood, 0.31 mg. per 100 cc. of blood; lead in urine, 0.38 mg. per liter; lead in spinal fluid, 0.17 mg. per 100 milliliters.

Blood chemistry: All normal; blood Wassermann, negative; spinal fluid pressure, first tap, 440 mm. of water; spinal fluid pressure, second tap, 300 mm. of water; spinal cell count, 5 lymphocytes per c.mm.; Pandy's test, 3 plus.

*Occupational History.*—Prior to this lead exposure this patient had worked for twenty-four years for the Nashville and Memphis Railroad. Upon coming to California he accepted employment at a Los Angeles pottery company where he worked for eleven months, sacking "frit." A study of the environmental factors was made at the author's request, while the patient was still hospitalized. The facts obtained were as follows:

At this plant the concern manufactures frit and grinds silica, clay, and other materials. Almost all of the material is for use in other plants owned by the Assured. Frit is manufactured by reducing silica, feldspar, lead oxide, boric acid, and a variety of other materials to a molten state in revolving furnaces. The molten material is quenched in water, emerging as a glassy substance, broken to fineness ranging from 4 to about 40 mesh. This material is then reduced to a fineness of approximately 200 mesh in large pebble mills and is removed manually with a shovel. Pebbles and other large pieces are removed with a coarse screen, hand agitated. The finished frit is sacked manually with the aid of a shovel and large funnel.

Supplementary report listing the various substances used in manufacturing frit is being prepared by the Assured. A number of different types of frit is manufactured, some containing lead, some not containing lead. Of the frit containing lead, concentration of lead ranges up to 70 per cent. A small amount of frit *not manufactured by the Assured is ground.* Sample of this material was furnished to the author.

O. B.'s principal occupation was unloading frit from the pebble mills and sacking same. Additional duties consisted of loading various sacked materials onto motor trucks, feeding silica rock into a jaw crusher with a wheelbarrow, and assisting in various labor operations. He did not work in the furnace room where the frit is mixed and melted. On occasion, he would load a dolly with the sacked unground frit in this department. Detailed report of all work performed by this employee is forthcoming from the Assured.

From this preliminary inspection, the principal exposure to substances containing lead seems to be in the mill unloading and sacking operation. This operation is very dusty and the workman is enveloped in a cloud of dust. Samples of dust-laden air from this point of exposure as well as other points of general exposure were taken, the dust count being tabulated below:

1. *Unloading frit from pebble mill and hand screening*
   Heavy concentration of visible dust; operation not exhausted
   Dust count: 27 million particles per cubic foot

2. *Filling paper sacks with frit*
   Considerable visible dust, operation not exhausted
   Dust count: 13 million particles per cubic foot

3. *General atmosphere following the sacking operation*
   Some visible dust
   Dust count: 7 million particles per cubic foot
4. *Loading sacks of ground silica (85 per cent 200 mesh) on hand truck*
   Visible dust
   Dust count: 3.5 million particles per cubic foot
5. *Piling sacks of ground silica in motor truck*
   Dust count: 2 million particles per cubic foot
6. *Dumping silica rock into jaw crusher*
   Dust count: 6 million particles per cubic foot

Employees are furnished respiratory protection in the form of M.S.A. Cumfo Respirators, Bureau of Mines approval No. 2101. The filter pad used in these respirators is manufacturers' serial number CR 9358, for lead dust. Although the management made some effort to enforce the wearing of respirators, the enforcement was not rigid. Since O. B. became ill, wearing of respirators is rigidly enforced.

In discussing O. B.'s personal habits with his foreman, the following information was obtained:

1. O. B. was very lax in wearing his respirator. Almost invariably the respirator would be hanging around his neck instead of in proper position. When cautioned, he would place it in position but would soon remove it. He was an inveterate smoker, but, as far as could be determined, he did not chew tobacco.

2. Lunch was eaten in the plant repair shop, a separate building located about fifty-five feet from the milling and sacking operations. Lunch is eaten here by most of the employees. The wash room is adjacent to the repair shop. The foreman did not know whether O. B. washed prior to eating his lunch.

3. Wash room facilities consist of stall showers, wash basin, toilet, and lockers. Showers and wash basin are provided with hot and cold running water. O. B. was not known to take a shower, although he would wash his hands and face at times prior to leaving for the day. He usually changed his clothes.

The Smith-Emery Chemical Laboratories analyzed the various samples of frit for their lead content, the following being typical examples:

| *No. 1 Frit* | |
|---|---|
| Feldspar | 11.7% |
| Silica | 4.4 |
| Lead silicate | 81.1 |
| Sodium nitrate | 2.8 |
| | 100.0% |

| *No. 2 and No. 34 Frit* | |
|---|---|
| Titanium oxide | 8.9% |
| Sodium oxide and potassium oxide | 6.3 |
| Calcium fluoride | 4.4 |
| Silica | 44.3 |
| Boron oxide | 6.2 |
| Lead oxide | 29.9 |
| | 100.0% |

| *No. 17 Frit* | |
|---|---|
| Rasorite | 4.5% |
| Soda ash | .7 |
| Fluorspar | 4.4 |
| Feldspar | 5.6 |
| Silica | 28.0 |
| Whiting | 1.2 |
| Litharge | 55.6 |
| | 100.0% |

| *No. 32 Frit* | |
|---|---|
| Sodium oxide and potassium oxide | 6.5% |
| Silica | 19.8 |
| Boron oxide | 14.5 |
| Lead oxide | 59.2 |
| | 100.0% |

| *No. 22 Frit* | |
|---|---|
| Boron oxide | 7.9% |
| Sodium oxide | 2.1 |
| Calcium oxide | 5.0 |
| Zinc oxide | 8.4 |
| Lead oxide | 38.4 |
| Silica | 32.8 |
| Aluminum oxide | 5.4 |
| | 100.0% |

*Comment.*—Following the second spinal tap this patient's mental condition improved rapidly and he was discharged from the hospital twenty days after admission. During the time of hospitalization and in subsequent periodic tests carried out at the out-patient department, a progressive decrease of lead in the blood and urine was demonstrated. Four months after the patient's discharge from the hospital the urine and blood were normal for their lead content. Three and one-half months after discharge from the hospital the patient took a job in a newsstand.

No attempt was made in this case to store lead or subsequently to delead the patient. *At no time was any calcium administered.* Aside from the two spinal taps, the only treatment given was for anemia, but that condition persisted for five months. It is worthy of note that this patient was followed for the next four years. During that time he was subject to several severe illnesses, one being a lobar pneumonia and the other septicemia. It was fortunate that during these periods urine could be collected for detailed analysis. It appeared that on both occasions the lead content was normal, proving that at least in this instance a severe illness had not released any stored lead.

CASE 4.—Lead intoxication from a misplaced decimal point. From 1911 to the end of 1928, L. W. worked in the lead industry in Chicago. During that time he did not engage in any process involving an actual lead exposure, for his work was that of repairing or installing machinery. During this entire period of employment he had no illness that in any way simulated lead poisoning, although he stated that on several occasions he experienced a "twitching of the muscles over the right shoulder blade." Likewise, during this period of employment he had never been "tested for lead."

In 1938, while living in Northern California, he suffered a paralysis of the right side of his face and tongue which, however, was of short duration. His physician at that time told him that "he was on the verge of a stroke." In 1944, while working in the hot sun in a small desert town in California, he suffered a cerebral hemorrhage with a hemiplegia. The company doctor in reviewing his occupational history noted that the man had worked in a lead industry and therefore intimated to the patient that the lead exposure might have caused him to have arteriosclerosis.

Soon after the cerebral accident the patient came to Los Angeles where he consulted his family physician. This doctor doubted the relationship of the previous exposure to lead to his present condition, but nevertheless sent the patient to a large and reputable hospital for a test of the lead in the whole blood. He received a report which read: "Lead in whole blood . . . . 0.14 mg./100 cc. Normal . . . . 0.01 to 0.06 mg./100 cc." This physician still felt that this was odd but informed the patient of his findings. A claim was made before the Industrial Accident Commission of California.

Shortly thereafter the patient was referred to the author, who went to the hospital to examine the laboratory record. The result of the test was written in the chemist's handwriting and read: 0.014 mg. per 100 cc. of blood. The stenographer, however, had made an error by placing the decimal point between the "0" and the "1," a most grievous mistake indeed!

*Comment.*—This case is an example of unnecessary litigation based upon two instances of false assumption. The first instance was that of thinking that a lead exposure of a minimal degree, if any, could have produced a cerebral hemorrhage to occur some sixteen years later. The second instance was to assume that lead could be circulating in the blood to the degree reported, after an absence from exposure of sixteen years. The case is a perfect illustration of thoughtless conjecture.

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# CHAPTER XXI

# CADMIUM

Cadmium has probably more lethal potentialities than any other of the metals. Lead, mercury, manganese, and many others may bring about an acute or chronic illness, but rarely do any of these cause sudden death from an acute exposure. Failure to appreciate the toxic properties of this metal has caused workers to be unwittingly exposed to its fumes and has furthermore allowed manufacturers to incorporate this metal in products without due warning of its presence.[1]

**Occupational Exposure.**—Cadmium does not only provide a fine protection against rust, but it takes a high polish so essential to certain alloyed products. It withstands excessive wear and has a relatively high melting point, qualities which make it invaluable in the manufacturing of bearings and other automobile parts. It is commonly used in electroplating, glass coloring, plating, soldering, and metalizing.

**Pathology.**—Because of animal experimentation, the reports have indicated a more extensive pathology than is probably found in the human being. Prodan[1] fed cadmium to cats to find subsequent involvement of nearly all of the viscera. But industrial cadmium poisoning results from inhalation, not ingestion. Legge[2] reported an autopsy on a workman exposed to the fumes of cadmium resulting from the melting of cadmium ingots. According to Legge, the autopsy on this man revealed inflammation of the lungs, gastrointestinal tract, kidneys, and spleen. In 1935, from two plants located in Ontario, fifteen cases of cadmium poisoning occurred and were reported by Bulmer, Rothwell, and Frankish.[3] Two of these men died and were autopsied, at which time the findings indicated predominant involvement of the lungs. According to these men, the post-mortem examination gave the following anatomic diagnosis:

Cadmium poisoning, edema, and congestion of the lungs (chemical), hemorrhages into the lung (chemical), partial collapse of lungs, proliferative interstitial pneumonitis (chemical), catarrhal bronchitis (chemical), cloudy swelling of the liver and kidneys, congestion of the spleen, and fatty infiltration of the pancreas.

Microscopic examination of the lungs showed congestion, hemorrhage, edema, alveolar collapse, desquamation of the lining cells of the alveoli, and marked proliferation of the lining cells of the alveolar spaces.

This report of the Canadian investigators indicates that the lungs bear the brunt of the assault from cadmium fume inhalation. Testimony to this is found in the autopsy of our patient, to be subsequently reported on in detail.

**Signs and Symptoms.**—The first symptom is usually dryness of the throat, or a sensation similar to sore throat. This is accompanied by or quickly followed by cough, and soon thereafter headache and dizziness will set in. After an interval varying usually from twelve to thirty-six hours, there occurs the characteristic feature of this type of toxemia; namely, severe, constricting pain in the chest and marked dyspnea. This delay is comparable to that seen in cases of phosgene poisoning, or nitrogen dioxide poisoning. After onset of pain in the chest and dyspnea, the respirations and pulse rate will increase.

An oddity encountered at examination is that auscultation of the lungs rarely reveals the degree of pathology indicated by the clinical picture. Until the patient is near death, râles are conspicuously absent and breath sounds may or may not be exaggerated. The x-ray picture will often show a surprising degree of widespread patchy bronchopneumonia throughout both lung fields, out of proportion to what one can hear in the lungs.

The signs and symptoms mentioned result from the inhalation of cadmium oxide. Ingestion of cadmium may occasionally take place but is rarely of industrial origin. Numerous cases have been reported where food, especially, it seems, lemonade, was prepared or stored in cadmium-coated containers and poisoning resulted upon ingestion of food or liquid. In such instances the initial and usually immediate sign, appearing within fifteen to thirty minutes, is severe epigastric distress accompanied by vomiting.

**Laboratory Tests.**—The cadmium solution may be precipitated by hydrogen sulfide as yellow cadmium sulfide which is insoluble in $(NH_4)_2S_X$ and soluble in hot dilute nitric acid. It is separated with Pb, Bi, and Cu from the remaining metals of the second group. Dilute sulphuric acid with $C_2H_5OH$ removes the lead, and $NH_4OH$ precipitates the bismuth as $Bi(OH)_3$, leaving the copper and cadmium in solution. If copper is present, KCN is added until the solution becomes colorless, when the cadmium is detected by the formation of the yellow cadmium sulfide with hydrogen sulfide. By passing in hydrogen sulfide, cadmium alone is precipitated as yellow sulfide, CdS, whereas copper remains in solution as potassium cuprocyanide, $K_4Cu_2(CN)_6$.

The present maximum allowable concentration of cadmium in the atmosphere is 1 mg. per 10 cubic meters of air.

**Disability.**—As previously stated, death may occur from undue exposure to cadmium fumes. Slight exposure will cause an illness lasting only a few days. Recovery from even severe exposure is complete, and no sequelae are encountered.

**Treatment.**—It has been suggested that since BAL is effective in the treatment of mercury and arsenic poisoning, it might also prove to be of value in cadmium poisoning. (For details of the use of BAL, see the chapter on Arsenic).

Drugs for Respiratory Relief.—The less seriously affected usually experience sensations similar to an upper respiratory infection, and measures commonly used for the latter often afford relief. These include the use of acetylsalicylic acid, 10 grains (0.65 Gm.), every four to six hours; capsules of acetylsalicylic acid, 5 grains (0.32 Gm.), phenacetin, 2½ grain (0.160 Gm.), and caffeine citrate, ½ grain (0.032 Gm.), at three-hour intervals; or capsules of codeine sulfate, ¼ grain (0.016 Gm.), and papaverine hydrochloride, ¼ grain (0.016 Gm.), at from four to six hour periods. A combination of ephedrine sulfate, ⅛ grain (0.008 Gm.), and amytal, ⅜ grain (0.024 Gm.), at from three to four hour intervals, may afford relief in some. Any of numerous cough syrups containing codeine phosphate or sulfate, pantopon, or dilaudid hydrochloride in combination with expectorants, such as ammonium chloride, may be of value. For example:

| | | |
|---|---|---|
| Codeine sulfate | 7½ grains | 0.45 Gm. |
| Ammonium chloride | 4 drachms | 16.00 cc. |
| Syrup of citric acid | 1 ounce | 30.00 cc. |
| Water to make | 4 ounces | 120.00 cc. |

Sig.: Teaspoonful every three to four hours.

Irrigations and Gargles.—Irrigation of the throat with warm salt and soda solution (a teaspoonful each of sodium chloride and sodium bicarbonate in one pint of water), or warm glucose solution ("corn syrup" one part; and water, two parts) every two hours may be undertaken. If irrigations are not possible, then gargling either with the salt and soda solutions, with 1:5,000 potassium permanganate, or with five aspirin tablets crushed in a glass of water may be substituted. Neither the use of silver preparations nor the frequent vigorous swabbing of the throat with other preparations is recommended.

**Inhalations.**—In some instances, inhalations of the following might prove soothing:

| | |
|---|---|
| Menthol<br>Camphor | āā 15.0 Gm. |
| Oil of Eucalyptus<br>Oil of dwarf pine needles | āā 30.0 cc. |

Sig.: One teaspoonful in a pint of water, steamed and inhaled.

Sedation in the form of barbiturates and intranasal drops may be needed, of from 0.25 to 0.5 per cent neosynephrin, or 0.33 per cent ephedrine in aqueous solution.

Oxygen Therapy.—Whenever a patient appears to be moderately or severely overcome by these fumes, placing him immediately within an oxygen tent is strongly recommended. This should be instituted without waiting for any signs of pneumonia for, as indicated by our fatal case, these may be absent. Furthermore, irrespective of the presence or absence of a pneumonia, oxygen therapy affords a relief for the air hunger which exists. If a specific organism is suspected to be present in a secondary invader, appropriate chemotherapy should be introduced.

**Case Histories.**—In 1941, the writer reported two cases of cadmium poisoning, which are repeated here as Cases 1 and 2. Since then he has seen three more cases of mild to moderate severity, but similar in all aspects to Case 1. In 1944 Spolyar[4] and associates presented an excellent report of five cases of their own, and at the same time brought the literature up to date. That report is recommended to the attention of the reader.

Case 1.—In June, 1940, a 24-year-old welder came to the clinic stating that on the day previously he had welded some "galvanized rods." His respirator was defective, but knowing that the job would be of short duration, he did not secure another respirator. Shortly after finishing this work he noticed an irritating cough, headache, and slight dizziness. That night he had chills, noted a dryness of the throat, the cough became more constant, and by morning he was aware of a heavy, oppressive feeling in his chest.

Examination revealed nothing unusual except increase in the pulse and respiratory rate. The temperature was normal; pulse, 90; respirations, 26; blood pressure, 116/78. The urinalysis and blood count were not significant, and the x-rays of the lungs were negative for evidence of any pathologic process.

Since the patient's illness resembled in some aspects a metal-fume fever, inquiry was made at the plant where he worked regarding the substance which he had welded. This author was informed that the rods were cadmium-coated. The patient returned to work six days after exposure, free of all symptoms.

The following case of the author's experience represents a contribution to the study of cadmium poisoning in that he was not only able to observe this case clinically, but secured complete laboratory studies, post-mortem findings, and microscopic slides of the various tissues of the body.

Case 2.—

*History.*—A 27-year-old Mexican laborer, employed by a metal company of Los Angeles, was referred to the author on April 14, 1941, with a diagnosis of metal-fume fever. Before proceeding with the details of this case, a defense of the physician who made the diagnosis of a metal-fume fever is justifiable and illustrates the confusion regarding cadmium poisoning, which was mentioned in an earlier paragraph. This doctor has for years treated workmen from this plant for the ordinary traumas incident to industry and has also treated numerous patients from this same plant for metal-fume fever and lead poisoning. He was never informed that cadmium was present in a new process used by this company. Furthermore, although a competent industrial physician, he told this author in discussing the case that his "reading material" had never mentioned the toxic effects of this metal. This digression well illustrates the fact that employers fail to realize that they expose their workmen to a hazard when cadmium is heated and that the physician has likewise not yet been impressed with the extreme toxicity of cadmium fumes.

While the patient managed to walk into the examining room, he was so exhausted that he was unable to speak for several minutes. He was obviously extremely ill, and after speaking a few words would stop to regain his breath. No ambulatory cardiac patient or even one suffering from a cardiac insufficiency of severe degree and confined to bed presented more extreme dyspnea. While this patient talked he clutched his upper chest. After considerable waiting, the following meager history was obtained.

Three days previously, April 11, 1941, he had crawled into a rotary furnace, thirty inches in diameter, to cut V-shaped openings from two longitudinal fins. (Details of his activities will be discussed in the comment on this case.) He wore no mask, and although the fumes caused him to cough, he finished the work, without stopping, in about forty minutes. Upon emerging from the furnace he was coughing considerably, but continued with other duties for the rest of that day. During the latter part of the afternoon his coughing became distressing, and he felt dizzy. That night severe headache set in, cough became constant, and during the night he became aware of a gripping chest pain. Sleep was impossible. He related that he would try sleeping in bed, then in a chair, then back in bed again. This stirring around increased the cough as well as the shortness of breath. The morning following exposure, the illness was reported to the employer, who sent him to the physician referred to previously.

The afternoon of the day following exposure, the patient's wife had him look into a mirror to see how blue his lips were. In spite of the administration of codeine, the chest pain increased in severity. The patient described this chest pain by closing his hands as if he were tightly squeezing something.

*Physical Examination.*—Inspection revealed this Mexican workman to be cyanotic and extremely dyspneic, with a facies that indicated he was suffering severe pain. His temperature was 98.4° F. upon admission; the throat was not inflamed; and in spite of a respiratory rate of 40 per minute, the lung examination showed evidence of no moisture or consolidation. The heart had a regular rate of 122 beats per minute, while the blood pressure was 140 systolic, 90 diastolic. The rest of the examination was not remarkable.

Twelve hours after admission, eighty-four hours after exposure, the temperature rose to 100° F., respirations remained at the rate of 40, the pulse was 130 beats per minute, and the lungs remained clear. Pantopon, one-third grain, did not relieve the chest pain. Unbearable headache accompanied the chest pain, as did dryness of the throat and cough upon the least exertion.

*Laboratory Examination.*—The initial laboratory findings were as follows:

Urine: specimen, voided; color, yellow; appearance, cloudy; specific gravity, 1.021; reaction, acid; albumin, 1 plus; amorphous deposits, none; pus cells, 1-2; mucous threads, numerous.

Blood: hemoglobin, 89 per cent; erythrocytes, 5,200,800; leucocytes per cubic millimeter, 14,800; polynuclear neutrophiles, 84 per cent; lymphocytes, 12 per cent; large mononuclears, 2 per cent; eosinophiles, 1 per cent; basophiles, 1 per cent; stippled blood cells, none.

*X-ray of Lungs.*—There were mottled increases in density throughout both lung fields. The pleural margins were clear. The cardiac shadow appeared to be normal in size.

On April 16, two days after admission to the hospital and five days after exposure, the temperature rose to 104° F., the pulse rate to 140, and the respirations to 50. The leucocyte count was 21,100, with 87 per cent polynuclear neutrophiles; the urine remained unchanged. A bedside x-ray film revealed multiple patches of increased density throughout the entire lung fields.

Worthy of comment is the fact that although this patient had been placed in an oxygen tent at the time of admission, it did not affect the respiratory rate. When under the tent he was definitely more comfortable and the chest pain less severe, but the breathing continued to be rapid and shallow, with movement confined to the area of the diaphragm. While the breath sounds were of the exaggerated bronchovesicular type, no moisture could be detected within the lungs.

For the next two days there was little change in the patient's general condition, except for a drop in the temperature and pulse rate, but the respirations continued to be excessively rapid. There was also no change in the lung findings, although the x-ray film showed widespread patchy bronchopneumonia. Thinking that a secondary invasion of the pneumococci might be present, sulfathiazole treatment was instituted, however without any effect. During all this time the patient remained perfectly lucid. But it became necessary to remove the oxygen tent momentarily, and immediately the patient would fight for air, toss to and fro, and appear frantic.

On the evening of April 19, nine days after exposure, the pulse rate increased to 160 and the respirations to 70, but the temperature did not rise until just before death. One hour before death the patient was mentally clear. During the last three days there was no chest pain, but cyanosis became more pronounced. On the morning of death, April 19, the bedside x-ray revealed the entire lung field to be overshadowed by widespread patchy pneumonitis, but there was no evidence of pleural fluid or solidary consolidation. On this last day, scattered râles were heard within the lungs, but there was nowhere any tubular breathing. The blood count and urinalysis taken on the day of death were as follows:

Urine: specimen, voided; color, yellow; appearance, slightly cloudy; specific gravity, 1.020; reaction, acid; albumin, trace; pus cells, 0-1.

Blood: hemoglobin, 90 per cent; erythrocytes per cubic millimeter, 4,500,000; leucocytes per cubic millimeter, 30,500; polynuclear neutrophiles, 88 per cent; lymphocytes, 7 per cent; large mononuclears, 2 per cent; eosinophiles, 2 per cent; basophiles, 1 per cent; stabs, 15.

During the height of illness a blood chemistry, blood Wassermann, and blood culture were made. The blood chemistry was normal, the blood Wassermann was negative, and the blood culture showed no growth. Sputum was never obtained for examination, since this patient's cough was nonproductive. A dark-brown urine has been reported as present in cases of cadmium poisoning. It was not so at any time in our case.

*Autopsy Report.*—The body was that of a well-developed and well-nourished man of approximately the age stated on the record, 27 years. There were no gross deformities. There were no evidences on the external surface of the body of any injury. The muscles were all well developed. The pleural cavity contained a thin, red fluid. The parietal pleura was smooth and glistening. The lungs did not collapse when the chest was opened. The right lung weighed approximately 900 grams. It felt solid. Frothy fluid escaped from the air passages when pressure was applied, but there were regions, particularly the lower lobe, in which a seropurulent fluid could be expressed from the smaller divisions of the bronchi. All of the small air passages appeared to be distended. The cut surfaces were quite wet. The visceral pleura, particularly of the lower lobe, was red and somewhat thickened. The left lung was similar, except that in the lower lobe there were regions up to 6 mm. in diameter, which were quite solid, from which seropurulent fluid could be expressed. Edema of this side was the same as that on the right.

The heart was not enlarged. All the valves were normal. No scars were found in the myocardium, but the muscle was somewhat flabby. Both right and left coronary arteries had common origin in the root of the aorta above the aortic cusps. Both arteries were patent throughout their extent. The liver appeared to be normal. The capsule was smooth and glistening, and the normal markings were present on the cut surfaces. The gall bladder contained black bile. There was no obstruction to the major bile ducts. The spleen was somewhat enlarged and soft, and the cut surfaces were a grayish-red. There were no abnormalities of the pancreas. Both kidneys were normal in size. The capsules stripped off easily. The cut surfaces were somewhat swollen and cloudy, but the normal markings were present. Except for slight grayish discoloration of the medulla in the lower pole, the right adrenal gland appeared normal. In the left adrenal gland, the medulla in the lower pole appeared to be replaced by gray-white tissue. This was of cartilaginous firmness. This tissue replaced less than one-sixth of the medulla and, except for this, the gland appeared to be normal. The urinary bladder contained clear, yellow urine. There were no abnormalities in the bladder. The mucosa of the stomach was intact. Some swelling and redness were present in the mucosa of the first portion of the duodenum. Except for this, there were no gross changes in the small bowel. The colon was normal. The gross appearance of the brain was normal.

From the gross examination, the cause of death in this case was bronchopneumonia, associated with marked edema of the lungs.

*Microscopic Examination.*—Sections of the following tissues were stained with hematoxylin and eosin:

(a) *Lung.*—The sections taken from the lower lobes of the lung showed that most of the alveoli were collapsed. There had been desquamation of the lining epithelium, and mingled with these cells there were numerous plasma cells and a few polymorphonuclear leucocytes. The interstitial tissue was thickened. The cells were swollen and in many places were separated by relatively large clear spaces. In other regions the tissue was quite compact. In the former regions there had been some infiltration with plasma cells and an occasional polymorphonuclear leucocyte. The alveoli which were open contained plasma cells, a few leucocytes, and large pale-staining round cells with centrally placed nuclei and pale-staining acellular debris. The lining epithelium of these alveoli was swollen and stained poorly.

(b) *Heart.*—There were no noteworthy alterations in the sections of the myocardium.

(c) *Liver.*—There were no alterations of the hepatic cells, and the lobular arrangement of the cells was intact. The capsule was normal.

(d) *Kidneys.*—Except for some swelling in the tubular epithelium there were no noteworthy sections of the kidneys. The glomeruli were intact, and the tubules were patent. There was no scarring.

(e) *Adrenal Gland.*—The sections had been taken through the lower poles of the adrenal glands. There were numerous, fairly thick strands of fibrous tissue replacing portions of the medulla. The medullary cells, however, appeared normal, and there were no alterations of the cortex.

## TABLE XIII — TABULATION OF SYMPTOMS OF CADMIUM POISONING AGAINST TIME IN HOURS, OF THEIR DEVELOPMENT

(Spolyar, Keppler, and Porter: J. Indust. Hyg. & Toxicol., 1944.)

| HISTORY | | | RESPIRATORY TRACT | | | | | GASTRO-INTESTINAL | | | GENITO-URINARY | C.N.S. | CARDIO-VASCULAR | GENERAL COMPLAINTS | | | PROGRESS | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Author | Number of cases reported | Exposed to | Duration of exposure | Irritation of throat | Cough | Dyspnea | Pain in chest | Nausea | Vomiting | Diarrhea | Urine | Headache and dizzyness | Pulse | Temperature | Chills | Weakness | X-ray follow up | Number of days to recover | Death occurred | Autopsy report |
| Sovet | 1 | Cadmium carbonate | 12–16 | 8–12 | | 72 | 8–12 | | + | | | + | Low 3rd day | | 72 | | | | | |
| | 2 | Dust from silver polish | 4–6 | | | | | | 6 | 6 | | 6 | | | | | | | | |
| | 3 | All 3 cases were servants | 4–6 | | | | | | 6 | 6 | | 6 | | | | | | | | |
| Stephens | 9 Cases Group | Cd. fumes from zinc smelting | | | | | + | + | | + | | | | | | | | | | |
| Teleky, Brezina | 1 | Cd. dust from punch press Opr. | | | | | | | + | | | | | | | + | | 24 | | |
| Legge | 1 | Cd. fumes from melting cadmium ingots to make paint | 2 | + | | | | + | | | Brown color | + | Rapid | | | | | | 4th day | Congestion, Larynx, Trachea, Bronchi, Stomach, Intestines |
| | 2 | | 3 | + | | | | + | | | Brown color | + | Rapid | | | | | | | |
| | 3 | | 3 | + | | | | + | | | Brown color | + | Rapid | | | | | | | |
| Schwarz | 1 | Cd. fumes, metal foundry | 5 | | 4–8 | 48 bron-pneu | 48 | 48 | | | | + | | | 48 | | | 1 yr. later dyspnea | | |
| Wahl | 1 | Cd. fumes | | | | | | + | + | | | | | | | | | | | |
| Fairhall | 1 | Cd. fumes in foundry | | | | | | | | | | | | | | | | | Few days | |
| Bulmer, Rothwell, Frankish | 1 | Fumes welding Cd. plated ware | 3 | 6–8 | 11 | 11 | 6–8 | | | | | | | | | | 20 mo. norm | 6 | | |
| | 2 | | 1.5 | | | 48 | 48 | 48 | 48 | | | | | 96–97 | | | | | 7th day | Pul. edema<br>Pul. hemorr. |

| | | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 3 | | 1.5 | | | | 24 | 10 | 10 | | | | | 100 increase on death | | | | | 8th day | Pul. edema<br>Pul. hemorr. |
| | 4 | Cases 2-15<br>Exposed to cadmium fumes from cupola Melting down 300 lbs. of cadmium plated rivets that were rejected | 1.5 | 12-24 | 48 | | | | | | | | | | | | | 11 | | |
| | 5 | | 1.5 | + | 72 | 72 | 72 | | | | | | | Norm. | | | | 11 | | |
| | 6 | | 20 min. | 4-8 | | | 4-8 | | | | | | | | | | | 11 | | |
| | 7 | | 5 min. | 4-8 | | | 4-8 | | 4-8 | | | | | | | | | 11 | | |
| | 8 | | Short | 8-12 | | | 48 | | | | | | | | | | | | | |
| | 9 | | | 3-4 | | | 48 | | | | | | | | | | | | | |
| | 10 | | | + | 48 | | 48 | | | | | 4-8 | | | | | | 11 | | |
| | 11 | | | 4-8 | 4-8 | | | | | | | | | | | | | 11 | | |
| | 12 | | | | | | + for 10 d. | | | | | 4-8 | | | | | | 6 | | |
| | 13 | | | + | | | | | | | | | | | | | | 3 | | |
| | 14 | (Trucker) | | | | | + for 11 d. | | | | | | | Norm. | | | | 11 | | |
| | 15 | (Trucker) | | | | | | | | | | | | | | | | 3 | | |
| Ohio indus. hyg. bulletin | 1<br>2 | Fumes-melting cadmium | Short | Soon | 4-8 | 24 | 18-24 | | | | | | | Mod. | +<br>+ | 4-8 | | 5 | | |
| Johnstone | 1 | Welding, cd. coated rod | Short | + | + | | 24 | | | | Norm. color. chem. | | Inc. | | | | | | | |
| | 2 | Fumes, cutting cd. with torch | 40 min. | Soon | | | 24 | | | | Norm. color 1 + alb. | 4-8 | 130-84H.<br>160-9 D. | 100-24H.<br>104-5 D. | | 72 | | | 9th day | Edema, lungs.<br>Kidneys, normal.<br>Brain, normal. |
| Spolyar, Keppler, Porter | 1 | Fumes from heating cadmium plated pipe with blow torch | 10 | 4 | 4 | 30 | 48 | 24 | 30 | | Norm. | 10 | 120-96H. | 99-96H. 106-death | 8 | 24 | | | 5th day | |
| | 2 | | 8 | 3 | 36 | 36 | 36 | | | | Norm. | 11 | | Norm. | 11 | 48 | | 7 | | |
| | 3 | | 4 | 4 | 24 | 24 | 24 | | | | Norm. | 4 | | | 8 | | 4 wks. norm. | 4 | | |
| | 4 | | 4 | 8 | 8 | 24 | 24 | | | | Norm. | 35 | Norm. | Norm. | 10 | 24 | 4 wks. norm. | 4 | | |
| | 5 | | 6 | 4 | 20 | 20 | 8 | | 20 | | Norm. | | | | | | 4 wks. norm. | 13 | | |

(f) *Duodenum.*—Sections cut through the first portion of the duodenum: The mucosa was intact and appeared normal in approximately half the section. It was absent down to the submucosa, and there was some round-cell infiltration along the remaining surface. The submucosal glands were normal, and there were no alterations in the muscular layers. The blood vessels were distended.

(g) *Brain.*—The brain contained no noteworthy alterations.

The manner in which this patient received the cadmium intoxication is not only interesting but instructive. The plant where this workman labored had been engaged for a number of years in reclaiming metal, chiefly lead, antimony, and tin, from discarded scrap metal. During 1940 they noted that another metal was present in the recovered substance, which upon analysis proved to be cadmium. They, therefore, devised a rotary furnace into which the scrap was thrown to be "sweated." This rotary furnace was thirty feet long and thirty inches in diameter. Within the furnace and on opposite sides, extending the length of this tubular furnace, were two fins which projected about four inches from the walls, the purpose of which was to tumble the scrap as the furnace revolved. It was decided that the recovery process could be made more efficient if V-shaped openings were cut at intervals in these two fins. Into this narrow, confined space went the Mexican workman without a mask to cut with an acetylene torch the fins coated with cadmium residue. The victim did not know that he was going to volatize cadmium, and had he known he would not have appreciated its dangers. The superintendent admitted he was not aware of the toxicity of cadmium, but contended that all the workmen are instructed to wear masks whenever using an acetylene torch to cut metal. Had someone been present to enforce that ruling, this death would not have occurred.

When this patient first presented himself with a diagnosis of metal-fume fever, it was felt that the diagnosis was erroneous because of two factors: (1) the complaint of severe chest pain and (2) the presence of extreme dyspnea. There is no acute industrial metal poisoning, to the writer's knowledge, which results in such distressing conditions. Just why they are present in severe cadmium poisoning is not quite clear, but the author believes it can be explained upon the basis of anoxemia. The immediate reaction to the fumes is apparently an acute inflammation of the respiratory tract so intense and so extensive that in order to lessen pain, massive splinting of the lungs occurs similar to that in pleurisy, but more marked. As the diffuse, patchy bronchopneumonia spreads and edema obliterates the alveolar spaces, the available area for aeration decreases until the blood receives no further oxygen. Successive x-ray films of the lungs of this patient revealed this gradual diminution of available normal lung space. Likewise, the autopsy showed the distention of the first portion of the bronchial vessels which accounts for the bronchovesicular breathing heard through the upper chest. The plugging of the alveolar spaces, particularly in the lower two-thirds of the lungs, explains why râles were not prominent. Both the autopsy findings and the microscopic study indicate the chemical rather than the infectious nature of the inflammation.

In a cadmium recovery room[5] a red-hot ash ignited cadmium dust on the floor which soon became a burning mass, from which arose yellowish-brown smoke of cadmium oxide. These fumes affected twenty-three persons by causing an initial irritation to the upper respiratory tract. After a period of delay, there occurred precordial constriction, dyspnea, and prostration. No fatalities occurred.

The five workmen who suffered from cadmium poisoning reported by Spolyar and associates[4] had been engaged in a process known as "flanging" in a small plant which had been doing this work for six months without any ill effects to the workmen. On Oct. 26, 1943, this plant was supplied by a prime contractor with pipe that looked somewhat different from the pipe which had been in use until then. However, in a day of continual changing of specifications, the management thought it was probably a change in the steel specifications and proceeded to flange this new pipe. In this operation the pipe was heated to a cherry red, at which time a hot wedge was inserted and the pipe flanged. Soon after beginning this work the men noticed more smoke than usual and in addition noted a dense yellowish-brown fume being emitted from the pipe. The first two workmen who began the operation became ill after four hours of exposure and were replaced by two other workers (Cases 3 and 4). In the next two case reports, which are Spolyar and co-workers',[4] the references in the case histories are to his series of five cases, not to the preceding cases reported by this author.

"CASE 3.—L. L. On October 26, patient worked in same area as Case 5 and thus sustained a contributory exposure of cadmium for 4 hours. That evening he developed a sore throat and 'just didn't feel good'. He thought he was getting influenza. Next morning, October 27, he felt fair and reported for work at 1:30 P.M., at which time he was assigned to the flanging of the cadmium plated pipe because Case 1 did not show up.

"By 4:00 P.M. (8 hours total exposure) he became ill on the job and complained about shortness of breath, pain in the chest, nausea and headache. He was taken home and put to bed, but a physician was not called.

"On October 28 and 29, he complained of severe pain in his chest, progressive shortness of breath, violent coughing, chills and fever, as well as nausea and vomiting.

"October 30, four days after initial exposure, the family physician was called in and the physician reported the following: 'Ashen cyanosis, rapid heart, dullness in bases of both lungs, no rales were heard, severe non-productive cough, bringing into play all the accessory abdominal muscles, shortness of breath and a degree of temperature.' He was treated symptomatically. At 2:00 P.M., October 31, the physician was again called and on arrival found the patient dead (five days after initial exposure). The family informed the physician that the patient was 'fighting for breath,' and just 'burning up with fever' before he died. An autopsy was not done, because the family would not consent to this procedure. This patient had the longest exposure to cadmium fumes in that he was exposed for 8 hours."

"CASE 4.—L. G., on October 27 was given the job of heater when Case 3 did not show up for work. That afternoon he developed marked irritations of the nasopharynx and headache. Later that evening he complained of shortness of breath plus a distinct pain in his chest.

"By October 28 the shortness of breath and chest pain increased and, in addition, a nonproductive cough developed. By remaining in bed five days he was symptom free by November 1st. During the illness no other complaints than those stated were elicited. At no time was he seen by a physician. X-ray studies four weeks after exposure showed the chest to be normal."

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# CHAPTER XXII

# BERYLLIUM

Beryllium is a rather rare element, though widely distributed in small quantities in certain feldspars, and in mica. In a few localities it occurs as beryl, a beryllium aluminum silicate. The same material colored light green by traces of chromium is the gem emerald, or colored bluish-green by traces of ferrous iron it is aquamarine. Beryl is the chief source of beryllium and beryllium compounds used in industry.

Intoxication from beryllium suddenly appeared in the United States among industrial workers during the years of World War II. However, beryllium poisoning had been reported in Germany in 1933, in Italy in 1935, and in Russia in 1936. In 1943, van Ordstrand and co-workers[1] observed men suffering from this poisoning and in their report stated they were unable to find any reference to the toxicity of beryllium in the American literature. They were probably not cognizant that at about the same time Fairhall and associates[2] were making an extensive study of the toxicity of beryllium.

Since the first reported cases in America there have appeared several reports of illness or death due to this substance, or to an acid radical of beryllium. Frabroni in Italy coined the term berylliosis, while others have preferred to call it beryllium sarcoidosis. That this condition is the same as Boeck's sarcoid is questioned by many.

**Occupational Exposure.**—Garner, in discussing by letter a case to be subsequently reported by the author, stated that there does not seem to be any relationship between the degree of exposure and the occurrence of the disease; he states that a number of cases have been observed where the exposure has been minimal. There are many industrial processes in which an exposure to beryllium may exist. It forms an alloy with many metals such as copper, nickel, and aluminum. Alloyed with copper, it produces a very ductile material capable of being formed into various shapes and having the qualities of extreme hardness and resistance to vibrations. Beryllium copper is used in making springs and diaphragms for altimeters in airplanes. Beryllium glass is used to filter out stray or reflected electrons in x-ray streams, to form beryllium alginate for the manufacture of a textile fiber from seaweed. One source of intoxication from which have come several reports is the fluorescent lamp industry. Obviously the processes of crushing or extracting the ore offer a definite hazard

**Etiology.**—It has been held by some investigators that beryllium poisoning causes primarily a chemical pneumonitis resulting from the action of acid radical such as the fluoride or oxyfluoride. Fairhall and associates[2] suggest the possibility of hydrolysis of such salts as chloride or sulfate, in addition to the fluorides and oxyfluorides. In a letter to Dr. Reginald Smart of Los Angeles, Dr. Leroy Gardner stated:

> "It is my impression that beryllium compounds probably facilitate the growth of some organisms which, in turn, cause the disease. We have grown acid-fast diphtheroid from the sputum of several cases and also from the blood of one of them. I don't like diphtheroids as a class. I am skeptical about this one, but it is the only clue we have to date. We have not been able to make it do its stuff in experimental animals. In spite of two years intensive work, I still lack a definite answer."

Again, in a subsequent letter to Dr. Smart, Gardner wrote regarding a case under study:

> "I believe it would be very profitable to have cultures of his sputum on blood agar in order to ascertain whether he has any of the peculiar, partially acid-fast diphtheroid organisms that we have seen in other cases. The growth of these organisms is very slow, and colonies do not appear until ten to fourteen days. At that time some very fine dewdrops appear which are barely recognizable as colonies. On smear, acid-fast or partially acid-fast forms are found."

Again, in writing of this problem, Gardner stated:

> "Certainly we have no reason to believe that tissue reaction is directly proportional to intensity or duration of exposure. Only a few cases have been employed in positions where there was apparently any real opportunity for inhaling fluorescent powders. In the majority of instances the contacts were casual, intermittent and of brief duration. In some of the industries using this process (fluorescent tube industry) there have been no cases whatsoever. This latter aspect of the problem makes it essential to postulate some other precipitating factor to initiate clinical disease."

**Pathology.**—Ulcerations and irritation to the skin have been observed, but the most interesting and serious pathology is within the lungs. As part of the discussion of the etiology the views of Gardner were presented. In 1946, Fairhall wrote:

> "The inhalation of beryllium sulphate dust was shown to be injurious to guinea pigs (67 per cent mortality in one exposure) in experimental work reported by Hyslop et al. in 1943; but when the more neutral potassium beryllium sulphate was substituted the animals were able to tolerate much larger doses (no mortality in large doses daily for 7 days). Later experimental work, using rabbits as experimental animals, has confirmed this earlier finding.
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> "Beryllium sulphate is so extensively hydrolysed that hydrogen is evolved when zinc is placed in the solution of the salt. It is understandable that the inhalation of beryllium sulphate dust would prove strongly irritating owing to the local production of sulphuric acid in contact with the delicate tissue of the lung substance. The production of a pneumonitis in the victim exposed to such irritating material is readily understood.
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> "Potassium beryllium sulphate, on the other hand, is soluble in cold water, is nearly neutral in reaction, and yields all the reactions characteristic of the

beryllium ion. It is not bound as a complex which might be expected to have different properties from other beryllium salt solutions. If beryllium were a protoplasmic poison, both beryllium sulphate and potassium beryllium sulphate should show similar effects."

Very few autopsied cases have been reported, but Hardy and Tabershaw[3] had an opportunity to study seventeen cases of workers in the fluorescent lamp industry, and Case 9 of their series is herewith partly reproduced:

"The pleural cavities contained about 50 cc. of pinkish blood-tinged fluid. The liver extended 3.5 inches below the costal margin in the right midclavicular line and 3 inches below the xiphoid. The diaphragm on the right rose to the level of the 4th rib and on the left to the 5th rib. The spleen extended about 3 inches below the costal margin in the left midclavicular line.

"Lungs: The lungs weighed 2400 grams. The lungs were large and voluminous and protruded forward and medially in the anterior mediastinum so that pleural surfaces were almost in apposition. The surfaces were reddish-pink and there were patches of fresh fibrin on the visceral pleura. Crepitus was almost wholly absent. The cut surfaces were dry, firm, and pinkish-red. There was no hyperemia of the vessels. In scattered peribronchial areas there appeared to be patches of darker red color which were moderately moist. The bronchial mucosa was reddened and the lumen contained a stringy whitish to yellowish mucus. The hilar nodes were diffusely but discretely enlarged. The larger nodes measured 1 x 2 inches and were rubbery in consistency and reddish in color on the cut surfaces. The pulmonary arteries showed a moderate distribution of flat atheromatous plaques.

"Heart: Weighed 260 grams. The right atrial and ventricular musculature was markedly hypertrophied and moderately dilated. The right venticle ranged in thickness from 4 to 8 mm. The pulmonary ring was moderately dilated but the valve appeared to be competent. The valve leaflets and cups were thin and translucent. The left side of the heart was not hypertrophied. The coronary vessels were thin and patent.

"Liver: Weighed 1500 grams. The liver was brownish-red in color and moderately firm. Cut surfaces showed a nutmeg type of lobular marking associated with marked hyperemia. The biliary ducts and the gall bladder were not remarkable.

"Spleen: Weighed 400 grams. The spleen was firm and the capsule thickened and tense. Cut surfaces were firm and appeared fibrous. There was marked hyperemia with a thin red pulp.

"Pancreas: The pancreas was of the usual size, shape, and consistency. There was moderate hyperemia.

"Adrenals: Not remarkable.

"Gastro-Intestinal Tract: Esophagus, stomach, and intestines were not remarkable.

"Genito-Urinary System: The kidneys weighed 400 grams. The capsules stripped with ease. Cut surfaces showed normal renal markings with moderate hyperemia. The pelves and ureters were not remarkable. The bladder contained about 2 ounces of dark urine. The mucosa was moderately hyperemic.

"*Microscopic Examination.*—Liver: The lobular architecture is obscured and there is extensive replacement of parenchyma by an organizing granulomatous infiltrate. The distribution is largely in the peripheral portion of the liver lobule with infiltrating strands invading the structure widely and meeting with that of contiguous areas. There is some proliferation of the bile ducts. The remaining liver tissue is largely confined to that about the central vein. The liver cords are atrophic and the sinusoids are dilated. There is hyperemia of the Kupffer cells. The cytoplasm of the liver cells is finely granular and contains some large and clear vacuoles. The nuclei in some instances are pyknotic and show karyorrhexis. Small hyaline cystic droplets are occasionally encountered. There is no evidence of regenerative changes.

"Lungs: Four sections. All sections show a diffuse fibrosis with coarse hyalinized strands of collagen among the alveoli and often in dense patches replacing alveoli. There are clear spaces in the interstices of the hyalinized material in which a cellular infiltration of lymphocytes, plasma cells, macrophages, epithelioid cells, and giant cells is diffusely spread without any particular arrangement. Alveolar walls are thickened and fibrosed, and many alveolar spaces are filled with a fibrous reticulum containing the infiltrating cells. Throughout all sections there is intense vascular engorgement with many dilated almost sinusoidal capillaries in the areas of fibrous proliferation. Sections including bronchus show tremendous vascular dilatation in the submucosa and about the cartilage plates. Bronchioles are not easily identified and have desquamated the lining epithelium. The pleura is slightly thickened and hyalinized and shows capillary dilatation and a moderate mononuclear infiltration. The large pulmonary vessels appear somewhat hyalinized in the externa, and some are surrounded by a loose collagenous reticulum.

"Hilar Nodes: Two sections. In both sections there appear to be a greater number of coalescent miliary nodules composed of a hyalinized collagenous reticulum containing giant cells, epitheloid cells and lymphocytes. The architecture of the lymph node is destroyed. There are a great many dilated capillaries throughout. Numerous areas are noted in which there is a very deeply staining hyaline fibrous material without cellular infiltration or vascular supply.

"Heart: Two sections. There is a marked fibroblastic intimal proliferation of the smaller branches of the coronary arteries.

"Spleen: One section: There is intense congestion of the sinusoids with red cells. The Malpighian corpuscles are small and the central arterioles show intimal thickening. Throughout the reticulum there are patches of hyaline fibrosis. The capsule and trabeculae are moderately thickened.

"Pancreas: One section. Marked hyperemia.

"Kidney: One section. Marked hyperemia. There is cloudiness of tubular epithelium and a granular fibrinous precipitate in the tubules. The arterioles are not thickened."

While this book was in the process of publication the author had a discussion with Willard Machle, of New York, who has had considerable experimental and clinical experience with the fluorides and more recently with beryllium. Machle believes that the lung pathology seen after exposure to beryllium is not due to fluorides or bacterial infection but can be accounted for by demonstrated action of beryllium alone, either as an oxide or neutral compound. The difference in the rate of onset of the disease may be explained by differences in rates of solubility of the compounds that are inhaled.

**X-Ray Findings.**—In a case which the author is reporting, a serial x-ray study of the lungs is described as follows:

The first films were stereoscopic (Fig. 32), taken July 6, 1944.* These reveal fine, reticular fibrosis scattered throughout both lungs, with a faint suggestion of very fine, miliary nodules. In addition, the bronchovascular markings in both hilar and perihilar regions, radiating into the periphery, are definitely accentuated. There appears to be slight calcification of the pericardium in the region of the apex. There may be a calcified nodule in the right base.

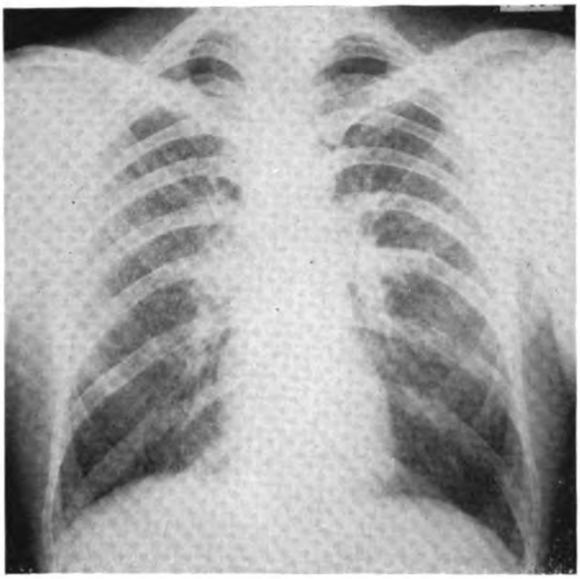

Fig. 32.—Film of worker exposed to beryllium which shows fine reticular fibrosis throughout both lungs with suggestion of fine nodules (July 6, 1944).

Films taken Aug. 22, 1944, show a slight increase in the pulmonary fibrosis previously mentioned and, especially, an increase in the bronchovascular exaggeration in the mid-lung fields. Films taken Nov. 2 and 9, 1944,† show a further increase of the bronchovascular accentuation and the suggestion of fine, miliary nodules previously noted is more definite. Stereoscopic films taken Nov. 8, 1944,† show very little change. Single films taken Jan. 8, 1945, and April 3, 1945, show a further increase in the accentuation of the bronchovascular markings throughout both lungs but more marked in the hilar and perihilar regions. The nodulation previously described is more definite.

Stereoscopic films taken May 7, 1945,‡ show faint miliary nodules scattered throughout both lungs but more marked in the mid-lung fields.

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* Taken by Dr. Edward Blaine.
† Taken by Dr. Carl Howson.
‡ Taken by Dr. Wilbur Bailey.

The accentuation of the hilar and perihilar structures seems to have decreased, but this is at least partly a matter of technique.

Stereoscopic films taken Nov. 25, 1945, at the California Hospital show very little change (Fig. 33). The fine stippling, or miliary nodulation, is quite obvious throughout both lungs. In the left apex, there appears to be some confluent infiltration. Stereoscopic films taken May 20, 1946, at the Hospital of the Good Samaritan (Fig. 34), show the very fine, miliary nodulation to be somewhat more definite. The linear and confluent infiltration previously noted in the left apex and first interspaces has increased slightly. There appears some linear infiltration of the right apex. It is noteworthy that the right cardiac border is more prominent. There is also increased density in both hila and particularly on the left, where there appears to be some conus fullness.

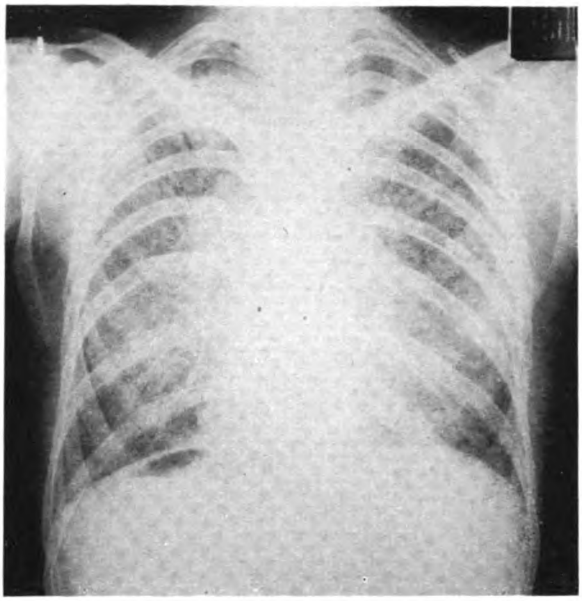

Fig. 33.—Film of same worker referred to in Fig. 32 taken thirteen months later (Oct. 25, 1945). Shows widespread, very fine nodulation with some confluent infiltration of left apex.

To the previously described films it can be added that Sosman and Wilson,[4] who studied the films of twelve among seventeen of Hardy and Tabershaw's[3] cases, offered the following opinion. They state that the roentgenologic appearance falls into three stages.

"Stage 1: The earliest recognizable variation from the normal is a fine diffuse granularity, presenting a fine sandpaper appearance, which under the magnification of a reading glass suggests a sand storm. There are no increased linear markings, no nodules, no coalescent lesions, no pleural thickening and no pleural effusion. The appearance is not the smooth homogenous ground-glass appearance of pulmonary edema or consolidation, but distinctly particulate in appearance. It is uniform and diffuse, extending to the periphery and includes the apices, although at first glance the apices seem to be spared. The hilar vascular shadows are usually normal and distinct in this stage.

"Stage 2: The second stage is characterized by a diffuse reticular pattern on the granular background. The hilar vascular shadows become fuzzy and indistinct and slightly enlarged. Only three showed enlarged hilar nodes, two of them moderate in degree, one rather marked.

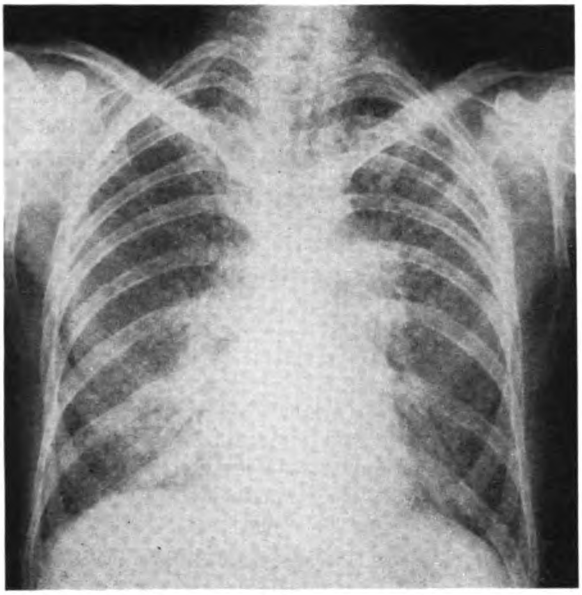

Fig. 34.—Film of same beryllium worker referred to in Figs. 32 and 33 taken seven months after film shown in Fig. 33 (May 20, 1946). For description, see text.

"Stage 3: Distinct nodules appear uniformly through the lungs, varying from 1 to 5 mm. in diameter, and the appearance now resembles a snow storm. Several in this stage have shown multiple small dark areas between the reticulo-nodular shadows, giving the appearance on the single film resembling the cut surface of a sponge. These could be due to small areas of emphysema. The nodules are evenly distributed throughout both lungs. They do not coalesce, do not calify or cavitate, and there is no definite linear fibrosis. The hilar shadows are quite fuzzy and indistinct, probably in part at least due to surrounding and overlapping nodulation. Pleural effusions are still absent, but the heart shadows in this stage become slightly larger and the pulmonary artery may become quite prominent. There is no basal emphysema demonstrable, although one patient's films showed progressive upward displacement of the hila and the interlobal fissure on the right side.

"The terminal picture may be complicated by heart failure (pulmonary congestion, hydrothorax and cardiac dilatation), but we saw no x-rays in this stage.

"One patient apparently did not go beyond Stage 1 and then showed subsequent clearing but did not return to normal. The granular appearance is still recognizable. One other patient improved roentgenologically after reaching Stage 3 (early). The hilar shadows remain enlarged, and the lung markings are definitely exaggerated, but the nodularity and the granularity have disappeared.

"The roentgenological appearance in the third stage may be closely simulated by sarcoidosis, or lymphangiectatic carcinomatosis and an occasional case of erythema nodosum with marked pulmonary changes. It is less closely simulated by acute silicosis, miliary tuberculosis, a rare case of diffuse fungus infection, and occasionally by the diffuse pneumonitis and miliary atelectasis which may follow an acute virus infection."

**Signs and Symptoms.**—Weakness, shortness of breath, and a cough which at first is usually nonproductive are the initial complaints. Later on, extreme nervousness, loss of weight, increasing dyspnea with cyanosis become evident. Hemoptysis may be present.

**Differential Diagnosis.**—Beryllium poisoning must be differentiated from miliary tuberculosis, silicosis, and Boeck's sarcoid. The occupational history in each case is obviously very essential to the diagnosis.

**Treatment.**—There is no specific treatment, and all drugs such as penicillin or the sulfonamides appear to be of no avail. General supportive measures should include the use of oxygen for those markedly dyspneic or cyanotic.

**Case History.—**

W. G. M. was first seen by Dr. Reginald Smart of Los Angeles on May 19, 1946, with the complaints of (1) increasing weakness for three years; (2) shortness of breath two and one-half years; (3) cough, two and one-half years; (4) loss of weight, three years; (5) anorexia, two years.

*Onset and Course of Present Illness.*—In January of 1944, the patient noticed palpitation and shortness of breath, especially on exertion. This gradually became more severe, so that he was short of breath all of the time and was only partially relieved by the administration of oxygen by nasal catheter. About the same time, he developed a dry cough, which later became productive of gray, greenish sputum. He progressively lost weight and had been unable to work since May, 1944. In July, 1944, he had a chest film made* which was diagnosed as pneumoconiosis. Since this time, the symptoms became progressively worse, so that he was now dyspneic all the time and, up to the time of my examination, had lost forty pounds. He had been examined by many physicians and was under the care of Dr. Carl Howson for a number of months.

*Inventory of Systems.*—Head, ears, eyes, nose, throat, and neck, no complaints. *Cardiorespiratory:* dyspnea, cough, and palpitation, as noted above. There had been edema of the ankles, more marked on the left, especially if he stood on his feet any length of time, for the past six months. He had had pleuritic chest pain and had an episode while in the hospital. This seemed to be aggravated at the times that he was more dyspneic. *Gastrointestinal:* no abdominal pain, nausea, or vomiting. The patient had diarrhea for two years, which was not entirely relieved by bismuth, kaopectate, or paregoric. This has not troubled him recently. *Genitourinary:* no dysuria, pyuria, or frequency. *Neuromuscular:* patient sleeps very poorly, due to the marked shortness of breath and the chest pain. Outside of the edema mentioned, there has been no trouble with the joints and no hyperesthesia or paresthesia.

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* Taken by Dr. Edward Blaine.

*Past Medical History.*—No significant illnesses previous to the attack of the strep throat and so-called influenza in 1943.

*Surgical History.*—Tonsillectomy at the age of 12 years. Right herniorrhaphy at the age of 18 years. Rectal surgery, which was done in 1945. No accidents.

*Family History.*—The patient's father died at 67 years of age of a stroke in 1944. His mother was 66 years of age and in good health. Two brothers and one sister were living and well. There was no known tuberculosis or other chronic pulmonary disease in the family. *Marital history:* wife, aged 47 years, was living and in good health; two sons, aged 6 and 8 years, were in excellent health.

*Occupational History.*—After graduating from high school in 1923, he worked for the C. W. Co. for two years; he then worked for the Bureau of Chemistry of the same company for twelve months. He came to California in 1926 and worked as a chemist in the neon sign industry, being first employed by N., Inc., and was with them until 1935. Later, he did glass blowing and bending of neon tubes. He worked for a number of neon sign and fluorescent lighting companies until January, 1942. On May 19 of this year, Mr. M. told me that he had some exposure to fluorescent powders and stated: "We began experimenting with fluorescent powders in the spring of 1938." He stated that sometimes the tubes came already coated with powders and it was his job to bend these tubes into different shapes and, in other instances, the tubes were coated where he worked by the application of fluorescent powders. He further stated that while his exposure to these powders was not intense, frequently some of the material would spill onto the bench or escape into the atmosphere.

In January, 1942, this man went to work for the C. S. Corporation where he was employed in the electrical maintenance of ventilation on ships that were being built or repaired. It was his job to keep the blowers operating. These were used to provide better ventilation in the holds of ships, double-bottoms, etc. In connection with this work, he was subjected to considerable smoke and dust. The fumes often consisted of arc welding fumes and other times of smoke from burners that were used to heat rivets. He was also subjected to asbestos dust and to iron oxide, or rust, that was being scaled off preparatory to painting. He was frequently subjected to particles of glass wool. This material was used extensively as an insulator in the refrigeration system.

It was in the fall of 1943, while working with this heavy dust, that he noticed difficulty in breathing, a choking sensation, and shortness of breath, particularly on climbing up flights of stairs to the top deck. There was also tachycardia.

The applicant's testimony before the Commission gives a more detailed description of his contact with the fluorescent powders. Although he never actually handled the powders, it was his job to blow into the open end of a tube containing the powders during the time that the glass was being heated and bent. It is entirely possible that the high temperatures necessary to bend the glass would cause the air to expand in the tube and even volatilize the chemicals used. Therefore, while the patient was not aware of any powder in the atmosphere which he breathed, it is more than likely that he did inhale these fluorescent chemicals during the three and a half years that he was working with them.

*Laboratory Work.*—*Urinalysis* done on May 20 and May 27, 1946, were negative, except for a faint trace of albumin. *Blood count,* taken May 20, 1946, was as follows: hemoglobin 20.1 Gm., 119 per cent; erythrocytes, 7,000,000 per cubic millimeter; color index, .85; leucocytes, 8,100 per cubic millimeter; polynuclear, 64 per cent; neutrophiles, segmented, 61 per cent; nonsegmented, 3 per cent; basophiles, 3 per cent; lymphocytes, 30 per cent; monocytes, 3 per cent; stained smear appeared normal; sedimentation rate, 1 mm. in one-hour Wintrobe. The packed cell volume was 61 per cent. The delayed sedimentation rate was, of course, affected by the polycythemia. It is noted that the packed cell volume was 61 per cent, as compared with the normal of 45 per cent to 47 per cent. Therefore, no conclusion can be drawn regarding the true sedimentation rate.

The patient was given penicillin intramuscularly and sulfadiazine by mouth. The latter drug was stopped after forty-eight hours because of a febrile reaction. The *blood determination* done on May 27, 1946, was as follows: Blood level at 2:15 P.M., sulfadiazine: 3.7 per cent. Chemotherapy was given in an effort to reduce the pyogenic organisms found in the sputum. As the sputum reports will show, there were many *Streptococcus viridans* and other pyogenic organisms found. *The vital capacity test* of May 21, 1946, is as follows: Maximum reading, 2,400 cc.; average reading, 2,250 cc.; normal for patient, 4,200 cc. Vital capacity: 57 per cent of normal. *Blood cultures:* May 22, 1946—Blood culture taken May 20, 1946, showed no growth after forty-eight hours' incubation. June 4, 1946—Blood culture taken May 20, 1946, showed no growth after fifteen days' incubation.

*Sputum Examinations.*—May 21, 1946: 250 cc. of mucoid, purulent sputum, said to be a seventy-two hour specimen, was received May 20, 1946. The entire specimen was digested, neutralized, and concentrated. Stain for tuberculosis was negative. Sputum was cultured for tuberculosis. Seven fresh morning specimens of sputum were collected in sterile containers and were examined by wet mount and culture for fungi. In no instance were monilia found on direct examination. However, several of the cultures showed a growth of a fungus, which at first was thought to be a monilia, but later studies suggested this to be a geotrichum.

*Physical Examination.*—*Chest:* Patient's breathing was very labored and he used the accessory muscles of respiration, to the fullest extent. There was some increase in the anteroposterior diameter. There was generalized emaciation of the muscles clothing the chest. Motion and excursion were symmetrical, but shallow and rapid. Resonance was moderately impaired over the upper third of both lungs. Breath sounds in these areas were vesiculobronchial and elsewhere they were bronchovesicular. A few fine, dry râles were heard after cough over both apices, anteriorly and posteriorly. *Heart:* Not enlarged to the left, or displaced. Rate, rapid, 100, but regular; no murmurs. There was, however, a 4-plus accentuation of the pulmonic second sound over the aortic second. *Abdomen:* There was a right herniorrhaphy scar. The abdomen was generally rigid and tense, due to the labored breathing. The liver was palpable two fingerbreadths below the costal margin but was not tender. Spleen not palpable. No palpable masses. No free fluid. *Extremities:* There was moderate, longitudinal curving of the nails and the nail beds were moderately cyanotic. This cyanosis was only partially relieved by the administration of oxygen by nasal catheter continuously. There was pitting edema of both feet and ankles and this was more marked at the end of the day and was relieved after oxygen had been administered for forty-eight hours.

*Progress in Hospital.*—May 19, 1946: This 39-year-old man entered with a two-year history of gradually increasing weakness, dyspnea, and cough productive of green and gray sputum. He had lost forty pounds (150 to 110 pounds). Physical examination reveals an emaciated, cyanotic, dyspneic, and orthopneic man, appearing chronically ill, with pulse 110 and respirations 50. The upper chest was dull to percussion, both anteriorly and posteriorly, and the expiratory breath sounds were abnormally distinct over the same area, especially at the apices, and shading off downward. There were medium inspiratory râles low in the right lateral chest. The heart seemed enlarged to the right to percussion. P2 is loud, while A2 is difficult to hear. No murmurs. The abdomen was difficult to examine because of the patient's position, but it was believed the liver was down 3 fingerbreadths, and was slightly tender. There was pitting ankle edema on the left and very slight edema on the right.

May 20, 1946: Patient is still dyspneic and general condition is unchanged. *Circulation time and venous pulse:* venous pulse: 5 cm. of water—pressure R.U.Q. —increase 2 mm.—release—5 cm. reading. Circulation time: arm to tongue, 26 seconds (glucose); arm to tongue, 37 seconds (saccharine); arm to lung, 16 seconds (ether). Results show venous pulse within normal limits and increased circulation time through the lungs.

May 27, 1946: Patient stated that for twenty six hours he had no sputum whatsoever but thinks the sulfadiazine made him feel bad.

May 28, 1946: Patient ran temperature of 101.2°F. about midnight the previous night and had some difficulty breathing. In the A.M. he was quite tired and dyspneic—color unchanged to any great degree. Sulfa level 3.7 mg. per cent. Temperature reaction might have been due to sulfa.

May 29, 1946: Patient very dyspneic and coughed severly and expectorated mucoid sputum. Liver enlarged two fingerbreadths below costal margin. Pitting edema of the ankles. Cyanosis as before.

May 30, 1946: Patient rested better in A.M. Had a fairly restful night. Color was unchanged. Patient had been using oxygen via nasal catheter since preceding afternoon and seemed to get some relief.

May 31, 1946: Patient rested somewhat better, using continuous oxygen. Color only slightly better. When oxygen was stopped, nail beds became cyanotic in two to three minutes.

June 3, 1946: Patient had been much more comfortable while taking oxygen. However, the dyspnea and cyanosis were not entirely relieved. The liver was now palpable at the costal margin. There was no edema at the ankles. Chest examination showed labored thoracic breathing, with impaired resonance over upper one-third of both lungs. Breath sounds in these areas were vesiculobronchial. Elsewhere they were bronchovesicular. A few fine dry râles were heard over both apices. No râles elsewhere. Heart rate, regular. No murmurs. Patient was to go home and continue oxygen and digitalis daily.

*Electrocardiogram.*—An electrocardiogram was taken on May 20, 1946, and the following was the interpretation: Average rate, 111; sinus tachycardia; P-R Interval, .20 second; T Wave inverted in Lead III; S Wave present in all leads; S-T Interval depressed in Lead II and III; right axis deviation. Conclusion: Right axis deviation. Sinus tachycardia. This tracing was compared with a tracing taken by Dr. E. L. Armstrong on Mr. M. on April 30, 1945. The following changes were noted: The right axis deviation had developed since 1945. The depression of the S-T segment in Leads II and III was not noted in 1945. Neither was the diphasic T in Lead II and the inversion of the T in Lead III, noted in the 1945 tracing. However, the sinus tachycardia was present in 1945, the rate being 110. The electrocardiographic findings, as well as the increase in circulation time through the lungs, and the clinical picture of dependent edema, enlargement of the liver, and marked cyanosis and dyspnea are all compatible with the diagnosis of right ventricular failure, or cor pulmonale.

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4. Sosman, Merill, and Wilson, Stanley: ibid.

# CHAPTER XXIII

# ARSENIC

Arsenic occurs in nature in combination with many metals as well as with sulfur. All of the soluble arsenic compounds are poisonous.

**Occupational Hazards.**—Among the following workers, arsenic affects the skin chiefly:

Arsenic roasters
Artificial flower makers
Artificial leather makers
Bookbinders
Brass foundry workers
Briquet makers
Bronzers
Calico printers
Carpet makers
Carroters (felt hats)
Chargers (zinc smelting)
Colored-paper workers
Color makers
Compounders (rubber)
Copper foundry workers
Copper smelters
Curriers (tannery)
Cut glass workers
Decorators (pottery)
Dye makers
Electroplaters
Enamelers
Enamel makers
Farmers and gardeners
Feather workers
Felt hat workers
Ferrosilicon workers
Picklers

**Pathology.**—Pathology consists chiefly of a local irritation causing lesions of the skin, mucous membranes, and appendages. Acute poisoning, as in suicide, murder, or accident by ingestion, will cause irritation of the stomach as well as degenerative changes of the heart, liver, and kidneys. Chronic absorption may lead to changes in the nerve and muscle fibers.

**General Discussion.**—Arsenical poisoning in industry is caused by exposure to dust or solid compounds or to the gas, arseniuretted hydrogen or arsine. The latter is much the more serious. The solid compounds are white arsenic, $As_2O_3$, the acetoarsenite of copper or Paris green, lead arsenate, and calcium arsenate. Industrially white arsenic is recovered by roasting and subliming arseniferous ores or subliming the flue and baghouse dust in smelters which work up such ores. In the lead smelters of Utah and Colorado and in the copper smelters of Montana, mild arsenical poisoning is frequent, but serious cases are very rare.

Paris green and the two arsenates have a very extensive use as insecticides, and the danger in this use concerns not only the makers of the compounds, but also the sprayer. In England white arsenic is used, together with sulfur and caustic soda or potash, for a sheep dip.

and similar mixtures are used to preserve hides and skins of birds and animals.

Arsenical poisoning has occurred in Germany among men handling hides from South America which have been treated with arsenic, and there have been cases in England among men unpacking bird skins. Thompson[1] reported a case of arsenical poisoning with peripheral neuritis in a tannery worker and quotes a chemist as having found arsenic in eleven of forty-two samples of furs.

The poisoning that results from exposure to arsenic dust usually is local, very rarely systemic. Skin lesions of various kinds, dermatitis, eczema, ulcers, scleroderma, bronzing, trophic changes in the nails, loss of hair, inflammation and ulceration of the mucous membranes and eyelids, are the commonest results of contact with arsenical dusts. Thickening of the skin of the palms may be the first symptom noted. Delicate parts of the skin such as the lips or eyelids, where mucous membrane and skin merge, are especially sensitive; so are the parts that are kept moist and warm, as the scrotum and the axilla. A chronic inflammation with slow, painless ulceration of the mucous membrane of the nose, which results in perforation of the septum, is, according to Dunlap,[2] of the Anaconda Copper Company, a very common accident in arsenic workers. It causes little suffering and does not result in deformity, for the bony part of the septum and the base are not involved. Dryness of the throat, mouth, and hoarseness are characteristic of arsenic workers.

In poisoning with lead arsenate the symptoms are more likely to be those of lead than those of arsenic. This is a fact well known to men engaged in producing this insecticide, and among the reported cases lead is accused more often as the cause than is arsenic. Aub and associates[3] saw a case of double wrist drop of twelve years' duration with difficulty in walking for three years in a man who had used lead arsenate spray in forestry work for twenty-seven years.

Fairhall and Miller[4] studied for two years the effect on rats of the arsenates of lead and calcium and the carbonate of lead. Most of the kidney damage was caused by the lead; blood destruction was greatest with the two arsenates. There was less storage of lead than arsenic in the soft tissues. In the bones there was more storage of lead carbonate than lead arsenate, the arsenate radical apparently decreasing lead absorption or increasing its excretion.

The picture of generalized, arsenical poisoning from solid arsenic, as seen in exceptional cases in industry, is rarely severe. Neuralgic pains and multiple neuritis with motor palsy, slight in degree, are characteristic of these cases. The palsy is likely to affect the long extensors of fingers and toes, as does lead, but it is distinguished from the latter by the severe neuralgic pains and by the tendency toward a multiple, symmetrical neuritis. A very rare form of industrial poisoning involving the optic nerve was described by Moleen,[5] who saw a case of skin lesions and double optic atrophy in a man using arsenical insecticides.

Of 135 cases of poisoning by solid compounds of arsenic, discussed by Legge,[6] thirty-eight had gastric symptoms, thirteen had tremors or muscular cramps, six had peripheral neuritis; of the remainder, all had lesions of the skin, in six of which there were keratoses or epithelioma. He speaks of three cases of epileptiform seizures, a most unusual manifestation of such poisoning.

**Arsine or Hydrogen Arsenide.**—Much more dangerous is poisoning by the gas, arsine or hydrogen arsenide. This is a subtle and powerful poison with a rapid characteristic action, but since it is always a result of accidental exposure, as arsine has no use in industry, and caused by arsenic, which is not known to be present, the cases often escape detection. The gas may be given off in any process which requires contact between dilute sulfuric or hydrochloric acid and a metal, usually iron, lead, zinc, or antimony, and since there is a large number of processes in which such contact occurs, there are many possibilities for the formation of arsine, for it is only necessary that metal or acid be contaminated with arsenic. The essential factors are arsenic and nascent hydrogen, the condition necessary for Marsh's test. Such an accident may occur in so many different kinds of industries that Wignall[7] suggested a search for hydrogen arsenide fumes in all cases where a mysterious form of illness shows itself among the workers; at least the question should be faced as to whether there is any process which could possibly result in the production of these fumes.

The symptoms of this form of arsenical poisoning are those of a powerful hemolytic agent. They appear after an interval, sometimes a few hours, sometimes a day or two, according to the size of the dose, and are characterized by anoxemia, nausea, vomiting, pain in the epigastrium, headache, and dizziness. The urine is dark, from the color of tea in mild cases to a Burgundy red in the more serious. Albumin and casts appear later, and in serious cases the urine becomes scanty with complete suppression in fatal cases. Somewhat later than the changed color of the urine, jaundice appears, with pain and tenderness in the liver region. It is a hematogenous jaundice from the destruction of red corpuscles and the accumulation of pigment in the circulating blood. A blood count shows anemia, sometimes very marked, and usually a proportionate fall in the hemoglobin, or the color index may be low. If the patient recovers, the urine regains its normal color, but albumin may persist for some days, and long after the acute symptoms have disappeared the anemia still is demonstrable. Wignall[7] has found arsenic in the urine eight weeks after the accident. In fatal cases the patient may pass into a typhoid state with delirium followed by coma, and there may be a general bronzing of the skin. Hemorrhagic nephritis and hemorrhagic inflammation of the liver are found at autopsy. The mortality in industrial cases is something over 36 per cent.

One of the commonest sources of arsine is the cleaning of the sludge from tank cars or storage tanks which have held sulfuric acid, after they have been emptied and flushed with water. Strong acid does not attack the metal, but dilute acid does, and if there is arsenic in either acid or metal, the dilution with the wash water allows the acid to attack the metal, liberating nascent hydrogen. Other sources are the spontaneous decomposition of ferrosilicon, the use of a galvanized zinc-coated pail in dipping out acid sludge, the production of hydrogen for filling toy balloons by treating zinc dust with hydrochloric acid, reduction of nitro compounds to amino in dye works, acid pickling of arsenic-contaminated metals, cyanide process for recovering gold and silver, and work in chemical laboratories, the last probably the most important of all.

The latest source of arsine in industry seems to be cadmium production from zinc-cadmium ore which carries arsenic. Acid is used in the leaching of the finely ground ore, and cases of arsine poisoning have occurred.

**Differential Diagnosis.**—Arsenic poisoning must be differentiated from botulism, mercurialism, and certain poisons such as carbolic acid. In the latter, the burns on the lips or mouth and the carbolic odor are characteristic. Mercurialism is differentiated by the salivation and pains in the mouth. Botulism is confined mostly to a gastroenteritis of one or two days' duration.

**Tests.**—Arsenic may be found in the urine, feces, hair, and nails. In chronic arsenic poisoning, it remains for a long time in the hair and nails. It has been found in the hair of an employee two years after cessation of his employment. This substance may be recovered from the bones and other organs after death.

**Medicolegal Aspects.**—

TEMPORARY DISABILITY.—This varies but is usually short. Skin lesions may be resistant to treatment, resulting in a temporary disability of several months. Gastrointestinal symptoms clear up shortly after exposure has ceased.

PERMANENT DISABILITY.—When this occurs, it is usually from a peripheral nerve involvement. If it is present after a two-year period, the functional loss should be estimated, as in any other partial permanent disability.

**Treatment.**—In an attempt to combat the effect of arsenical gases, British scientists initiated a search which led to the synthesis of

2, 3-dimercaptopropanol, more familiarly known as BAL. There is now a convincing body of evidence[8] that the toxic effects of arsenicals are referable primarily to the fact that they combine with -SH groups in the tissues, and thus block one or more physiologic systems vital to the cellular economy. In a search for compounds capable of acting as decontaminants or antidotes for toxic arsenicals, Stockten and Thompson[9] believed that a dithiol compound might form a stable ring with arsenicals. Of the various substances tried, the most effective proved to be 2, 3-dimercaptopropanol.

To overcome the deterioration which occurred in aqueous or propylene glycol solutions, BAL has been incorporated in a peanut oil-benzyl benzoate solution. This is marketed in ampule form. Observations on the human being by Modell and co-workers[10] indicate that injections may produce burning or tingling of the nose, eyes, mouth, and skin, perspiration and a sense of warmth, sometimes pain in the limbs, jaws, abdomen and head, lacrimation, salivation, vomiting, unrest, and fatigue. The heart rate may be accelerated and both systolic and diastolic blood pressure levels elevated.

It is recommended that BAL be given intramuscularly, the dosage being adjusted to body weight, 2.5 mg. per kilogram, or 0.025 cc. per kilogram. In a man weighing 165 pounds (75 kilograms), the unit dose per injection is 1.8 cubic centimeters. This dose should be at four-hour intervals for a total of four to six injections for the first two days, and then reduced to once or twice daily until recovery appears complete.

Sulzburger, Baer, and Kanof[11] state that approximately 0.3 Gm. of BAL applied to the skin in the form of BAL 10 per cent in KY jelly or in petrolatum can be used on the skin with safety. Some have noted a burning for about one-half hour, or a transitory local erythema.

It is known that exposure of the eyes to arsenic vapor may cause lesions of the cornea with rapid tissue necrosis. To counteract this, a single instillation of a 5 per cent BAL solution or ointment has been recommended as being exceedingly effective.[12]

For those cases which have not had the benefit of treatment with BAL and where a peripheral neuritis has been a sequela, it is recommended that 20 mg. of synthetic vitamin $B_6$ in sterile physiologic saline be given twice daily combined with intramuscular injections of 50 mg. of alpha-tocopherol.

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# CHAPTER XXIV

# MANGANESE, MAGNESIUM, PHOSPHORUS, ANTIMONY, AND ZINC

## Manganese

Manganese is now considered to be indispensable in the production of steel. During World War II it proved to be the most critically scarce of all the metals in America's war effort. It is calculated that every ton of steel produced requires fourteen pounds of manganese to remove sulfur and oxygen. Pure manganese is a hard, brittle metal which very closely resembles iron.

Since manganese imparts strength and hardness to steel products, about 90 per cent of this metal is used to form alloys, the most important of which are ferromanganese, silicomanganese, spiegeleisen, and silicospiegel. Manganese also forms alloy with aluminum, tin, arsenic, bismuth, boron, copper, and zinc.

**Occupational Exposure.**—The minerals which serve as ores of the important heavy metals are usually found in nature in admixture with useless impurities, collectively known as gangue. The ore particles are heavier than the accompanying gangue and hence may be separated by crushing the ore to a fine powder. It is during this process that the workers are in great danger. Other uses for manganese are in the manufacturing of batteries, glass, pottery glazes, dyes, matches, and fireworks, in disinfectants, deodorants, and germicides, and in the bleaching of textiles.

**Pathology.**—Injury to the body is due to the chronic or prolonged exposure to manganese. It has a predilection for the central nervous system, and the ganglion cells of the basal ganglia are more commonly affected. Reports of microscopic examinations state that a considerable scarring and shrinking together with gliosis is generally noted.

According to Voss[1] the number of manganese poisonings is small in comparison with the number of exposed workers. He believes this is due to the fact that only a small number of persons have a predisposition for this poisoning; that not all manganese compounds are capable of exerting a toxic action; and that the danger of occupational poisoning occurs only where manganese compounds are inhaled or swallowed in adequate quantities for a comparatively long time. Voss observed a case of a man who had had a mild exposure for ten months and a severe exposure for seventeen months. Other men working under the same conditions developed no signs or symptoms. This patient showed a masklike face, amyostatic symptoms, disturbances in speech and gait,

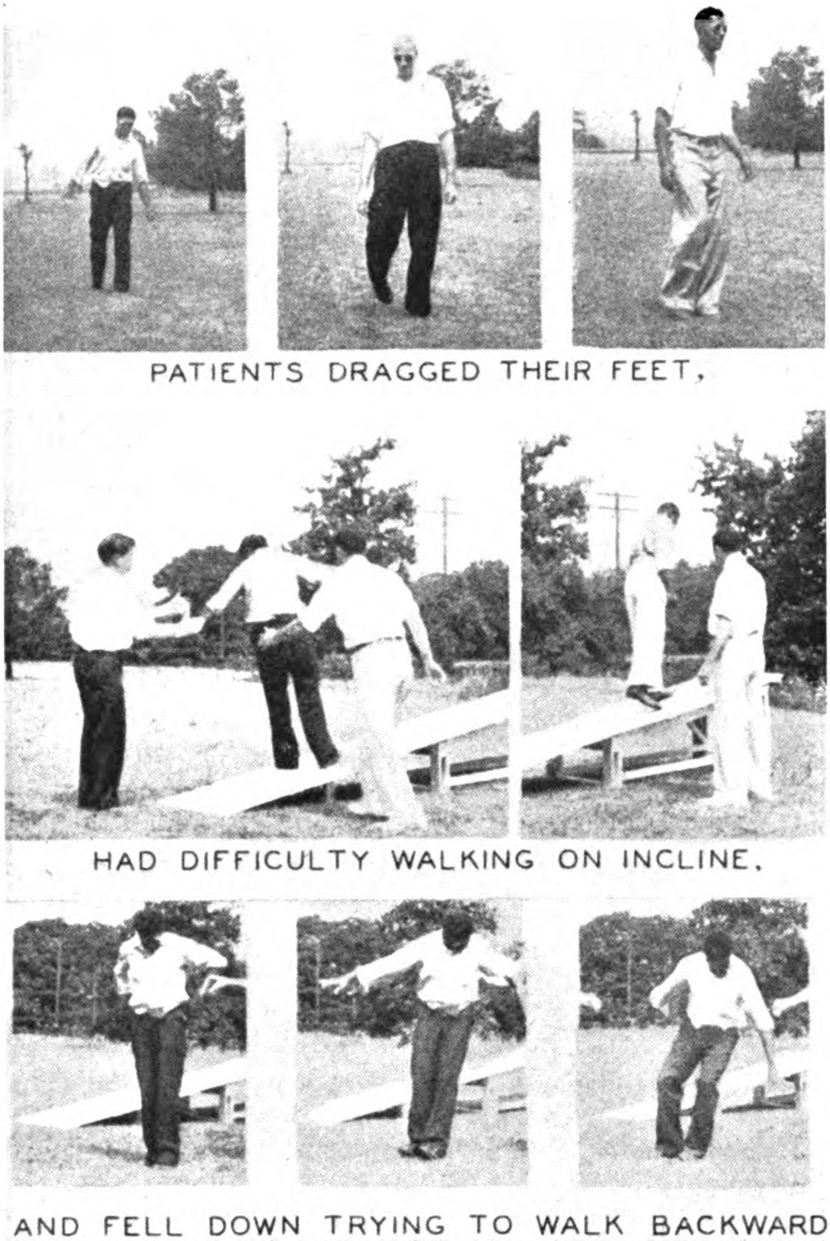
PATIENTS DRAGGED THEIR FEET,
HAD DIFFICULTY WALKING ON INCLINE,
AND FELL DOWN TRYING TO WALK BACKWARD

Fig. 35.—Patients with chronic manganese poisoning. Photographs taken with a motion-picture camera. (U. S. Public Health Service, Bulletin 247.)

micrographia, laughing, weeping, and stuttering. Stored manganese was indicated by the presence of this substance in the feces eight months after the cessation of exposure. Nervous lesions in severe poisoning usually remain constant or increase in severity, but the patient observed by Voss improved.

Bryan[2] reported a case of manganese poisoning in a 47-year-old man who sought medical treatment because of disturbance of gait. The initial symptom in three of six patients examined by Gayle[3] was a disturbance of gait. Canavan, Cobb, and Drinker[4] were fortunate to observe the autopsy findings in a 69-year-old man who died of cardiorenal disease fourteen years after the onset of symptoms resulting from this occupational hazard. This man had been exposed to manganese for four years. His symptoms had been disturbance of gait, stiffness of the legs, frequent falls, fatigability, and sleepiness. The chief autopsy findings were degeneration of the nerve cells, satellitosis, and gliosis in the basal ganglia. There was also widespread atrophy in various portions of the brain. The *Bulletin of the United States Public Health Service*[5] showed pictures of patients walking but dragging their feet, attempting to walk up an inclined surface with difficulty and falling down when they tried to walk backward. (Fig. 35.)

Croupous pneumonia has been reported by foreign observers as being fairly common. However, in a comparison of two groups of cases studied by the United States Public Health Service,[5] there appeared no significant variations in the incidence of pulmonary disease among the exposed and the nonexposed group. It was believed that manganese did not predispose to tuberculosis and that it did not cause pneumoconiosis.

**Effect on Blood.**—Extensive laboratory examinations of workers indicated that a low white cell count with a decreased percentage of neutrophiles, a slightly lowered blood calcium content, and a slight reduction in the middle zone of Lange's test were often associated with manganese poisoning. A lowered hemoglobin content was observed in several of the manganese workers. Analysis of the blood for manganese content was of no assistance in this study.

Urinary manganese appears to reflect a present or past exposure to manganese compounds rather than evidence of intoxication. Impairment of kidney or liver function was not observed.

**Absorption and Elimination of Manganese.**—In the discussion of the pathologic effects caused by manganese, it was stated that intoxication results from chronic exposure. The length of time which the illness requires in order to develop varies considerably. According to Fairhall and Neal,[6] most workers become sick within the first two years of exposure. In some patients, however, illness did not occur until after twenty years or more of exposure, while Edsall, Wilbur, and Drinker[7] reported symptoms in a worker after one and one-half months of work.

Intoxication is the result of absorption from the lungs rather than by ingestion. It is excreted in the urine and feces. But small amounts are stored in the various tissues.

**Signs and Symptoms.**—Languor, sleepiness, stolid, masklike facial expression, low monotonous voice, muscular twitching, cramps in the leg muscles, increase in tendon reflexes, ankle and patellar clonus, retropulsion and propulsion, slapping gait, and uncontrollable laughter are some of the signs and symptoms in manganese poisoning. The patient is usually aware of muscle stiffness and twitching first, and he then notices difficulty in walking.

**Differential Diagnosis.**—Paralysis agitans, multiple sclerosis, and progressive lenticular degeneration must be differentiated from chronic manganese poisoning. The occupational history is, therefore, important in initiating suspicion that one is dealing with manganese poisoning.

**Tests.**—There is no single laboratory finding which offers conclusive evidence of manganese poisoning. Manganese may be found in the

TABLE XIV. NUMBER AND PER CENT OF WORKERS, WHO REPORTED CERTAIN DISORDERS, IN ORDER OF FREQUENCY

(United States Public Health Bulletin 247, 1940.)

| IMPAIRMENT | PERCENTAGE | | | NUMBER OF CASES | | |
|---|---|---|---|---|---|---|
| | AFFECTED | EXPOSED, NOT AFFECTED | NONEXPOSED | AFFECTED | EXPOSED, NOT AFFECTED | NONEXPOSED |
| Total examined | 100 | 100 | 100 | 11 | 23 | 16 |
| Lassitude | 91 | 4 | 0 | 10 | 1 | 0 |
| Drowsiness | 91 | 4 | 0 | 10 | 1 | 0 |
| Gait disturbances | 91 | 0 | 0 | 10 | 0 | 0 |
| Dyspnoea, slight | 91 | 0 | 0 | 10 | 0 | 0 |
| Tremor of body or extremities | 91 | 0 | 0 | 10 | 0 | 0 |
| Muscular weakness | 82 | 4 | 0 | 9 | 1 | 0 |
| Muscular cramps | 82 | 9 | 6 | 9 | 2 | 1 |
| Speech disturbances | 73 | 0 | 0 | 8 | 0 | 0 |
| Sexual disturbances | 73 | 17 | 0 | 8 | 4 | 0 |
| Metallic taste | 64 | 0 | 19 | 7 | 0 | 3 |
| Vertigo | 55 | 4 | 6 | 6 | 1 | 1 |
| Paraesthesia | 55 | 0 | 6 | 6 | 0 | 1 |
| Palpitation | 55 | 0 | 6 | 6 | 0 | 1 |
| Anorexia | 45 | 4 | 6 | 5 | 1 | 1 |
| Muscular pain | 45 | 0 | 0 | 5 | 0 | 0 |
| Twitching of fingers | 45 | 0 | 0 | 5 | 0 | 0 |
| Headache | 45 | 35 | 19 | 5 | 8 | 3 |
| Joint pains | 36 | 4 | 0 | 4 | 1 | 0 |
| Increased perspiration | 36 | 0 | 6 | 4 | 0 | 1 |
| Salivation | 27 | 0 | 0 | 3 | 0 | 0 |
| Impulsive weeping | 27 | 0 | 0 | 3 | 0 | 0 |
| Impulsive laughter | 27 | 0 | 0 | 3 | 0 | 0 |
| Difficulty in swallowing | 18 | 0 | 0 | 2 | 0 | 0 |
| Violent temper | 18 | 0 | 0 | 2 | 0 | 0 |
| Nausea | 18 | 0 | 0 | 2 | 0 | 0 |

excreta, but this only indicates the fact that manganese has been absorbed. In suspected cases a low white cell count with a reduced percentage of neutrophilic leucocytes is a significant confirmatory aid. The group working under the supervision of the United States Public Health Service failed to find any significant changes in the bones upon x-raying a series of men poisoned by manganese, nor did they find any value in the study of blood analyzed for manganese. They did note that the blood calcium was lower on the average in the poisoned patients than in the nonaffected. A slight rise in the middle zone of the colloidal gold curve was thought to be noteworthy. These workers also noted a reduction in the middle tubes of Lange's test, similar to that observed in paralysis agitans. It was held that the Lange test may prove helpful in differentiating manganese poisoning from active multiple sclerosis.

TEST OF ATMOSPHERE.—In cases where a questionable history of exposure exists, it may be of advantage to test the atmosphere at the place of occupation. It is thought that 60 mg. of manganese per 10 cubic meters of air is a safe atmosphere for workmen.

**Treatment.**—

PROPHYLAXIS.—The prevalence of this disease can be limited by engineering control. Isolating the process under hoods or exhaust ventilation, control of the dust by wet processes, the use of mechanical conveyors, and examination of the atmosphere at intervals are all preventive measures. Quarterly medical examinations should be routine in all mills or places where manganese compounds are handled in order to detect early signs or symptoms. Mild neurological signs or symptoms, such as drowsiness, languor, muscular cramps, and twitching, perhaps coupled with an otherwise unaccountable low white cell count, indicate removal of the workman from the hazard. Daily showers following work should be encouraged by installation of shower baths and education of the employee in this regard.

Nothing of real value in the treatment of these patients has been described. For those mildly affected, much improvement, perhaps total, will occur on their removal from the exposure. For those severely affected, little improvement may be expected.

VITAMIN $B_1$ AND CALCIUM.—Well-rounded, adequate diets should be given these patients, and general tonics may be used. There is some question concerning the use of thiamine hydrochloride (vitamin $B_1$) since experimentally large doses of vitamin $B_1$ encourage retention of manganese, and hence $B_1$ might be *contraindicated* here. Since low blood calcium levels have been demonstrated by some, oral calcium preparations and milk may well be included in the diet. The patient should be encouraged to drink adequate amounts of fluids and should be given cathartics, if necessary, in order to secure adequate elimination.

SODIUM THIOSULFATE AND LIVER EXTRACT.—McNally[8] thought a patient of his showed some improvement while 15 grains (1 Gm.) of sodium thiosulfate in 10 cc. of aqueous solution were given intravenously every other day. He cites the feeding by Charles[9] of liver to these patients with what appeared to be improvement in some. When improvement under liver therapy was noted, it was rapid and occurred in the first few days or so of the treatment. The use of liver extract intramuscularly, then, in dosages of from 15 to 30 units a day for four consecutive days, and the repetition of this dose two or three times a week over an indefinite period of time, depending upon the individual patient, is suggested.

CASE 1.—"White male, aged 50 years.

"*Occupational History.*—This man quit working in the manganese plant in 1918, after having worked there for two years, running a pulverizing machine. He stated that he was grinding both chrome and manganese ore at that time, working inside the whole time. He stated that the dust conditions were particularly bad, and he did not wear a respirator. He was transferred out of this plant in 1918 because of diffiiculty in walking. Following this, he worked with the mixing pans in the brickyard for six years and after that running the pump and tending boilers in the brickyard for three years. He was assigned to this light work on account of partial disability. Prior to this industrial work, he had farmed for thirteen years and had been a mule tender in a woolen mill for three years. He had not worked at all since 1928.

"*Personal History.*—His wife was living and well. They had two children living. His wife had one miscarriage after influenza in 1927. He admitted being a light user of liquor and a rather heavy user of tobacco, mostly in the form of cigars.

"*Past Medical History.*—The patient stated that he had had frequent severe attacks of tonsillitis. He had typhoid fever at the age of 13 and was in the hospital for five weeks. He had neuralgia of the face years before, when his teeth were bad. He was operated on for hemorrhoids in 1930. He had a chronic, severe cough while working in the manganese mill, lasting about one and one-half years.

"*Present Medical History.*—Patient dated the onset of his present condition back to 1918, about the time he was transferred out of the manganese plant. It began with a slight weakness, which grew worse and still persists. Marked disturbances in gait, with stumbling, began to trouble him, with inability to lift the right foot. He was troubled with weakness and cramps in the legs, falling down on going backward, inability to turn around, and difficulty in going up and down hills, especially the latter. He noticed moderate drowsiness, which has persisted. Metallic taste and loss of appetite were noted. He had generalized tremor of the muscles, muscular weakness, especially of the right leg, and muscular cramps, worse at night, throughout both legs. He was troubled with numbness and tingling of the forearms, hands, feet and legs, which was intermittent, and a stuttering, halting speech. He had some difficulty in swallowing food.

"Since 1925 he had noticed short attacks of impulsive weeping. There had been no change in his sexual functions. His wife had had three pregnancies during this illness. Since the onset of these symptoms he had noticed a slight shortness of breath on exertion. He stated that he had noticed black spots and stars in front of his eyes at various times during the last twelve years. He had occasional slight attacks of palpitation of the heart. He had occasional twitching of the fingers and legs, mostly when sitting down. Several of these symptoms had been improving in the past few years. The trembling of the extremities, and muscle tremor in general, was improving, being intermittent in character and troubling him only occasionally at the time. However, the weakness in the right leg seemed to be getting worse. He believed his ability to talk was improved considerably.

"*Physical Examination.*—White male, aged 50, not acutely ill. Height, 71 inches; weight, 142 pounds; temperature, 97.4°F.; almost white hair; eyes and ears, essentially normal; moderate obstruction to breathing was present in the nose; rather marked pyorrhea alveolaris, a slight amount of dental caries, all upper teeth were out; a coarse, moderate tremor of the tongue was noted; neck, normal.

"(a) *Chest:* Girth at inspiration, 94 cm.; at expiration, 86 cm. The physical examination of the chest was essentially negative. The stereographic x-ray films of the chest were essentially negative.

"(b) *Circulatory System:* The examination of the heart was essentially negative except for some diminution of all heart sounds. Before exercise the heart rate was 80; immediately after exercise, 96; and three minutes after exercise, 80. The blood pressure before exercise was 124/84; immediately after exercise, 126/86. No peripheral arteriosclerosis was noted. The electrocardiogram showed a heart rate of 76; conduction time, 0.12 second; left axis deviation and low voltage, 0.45 millivolt.

"(c) *Abdomen:* Except for a left inguinal hernia, supported by a truss, the examination of the abdomen was negative.

"(d) *Extremities:* Examination of the extremities revealed atrophy of the left thigh and calf, the circumference of the left calf being 2½ cm. less than the right.

"*Neurologic Examination.*—Besides the atrophy noted in the left thigh and calf, there was a general diminution in power of all groups of leg muscles. The facial muscles were not affected, except for those of the forehead. Only tongue tremor was observed. The ankle jerks were slightly decreased. Both Achilles tendons were abnormally sensitive to compression. The Romberg test was slightly suggestive but not positive. The gait was spastic and slapping in character. He had a right foot drop, and had to swing the leg to walk. Retropulsion and propulsion were noted, with the subject falling down on walking backward. On the inclined plane he had marked difficulty walking up, owing to the right foot drop, and he fell down in walking down backward. Walking sideways was quite slow and tedious. There was no intention tremor in lifting the water glass. There was a slight stuttering observed with fast economical speech, indistinct at times. Masked facies was not observed. A specimen of handwriting revealed no intention tremor or micrographia. The examination of the skin was negative. The psyche was unaltered.

"*Laboratory Findings.*—(a) *Urine:* Specific gravity, 1.013; amber; acid; no albumin or sugar; 0.006 mg. Mn per 1; no casts; occasional red blood cell; 1 or 2 pus cells per high-power field.

"(b) *Blood:* Erythrocyte count, 4,900,000; 14.5 Gm. hemoglobin per 100 cc. (86 per cent); color index, 0.88; reticulocytes, 0.30 per cent; white blood count, 10,200; platelet count, 255,000.

"(c) *Schilling Differential Count:* Showed 53 per cent segmented neutrophiles, 39 per cent lymphocytes, 6 per cent monocytes, 1 per cent eosinophiles, 1 per cent basophiles.

"(d) *Coagulation Time:* 1 minute, 50 seconds; bleeding time, 3 minutes.

"(e) *Kahn Test:* Negative.

"(f) *Blood Chemistry:* (mg. per 100 cc.) Blood glucose, 93; total nonprotein nitrogen, 40; serum chloride, 366; serum calcium, 9.5; and serum phosphorus, 3.9.

"(g) *Cerebrospinal Fluid:* Clear and colorless; very faint trace of globulin; cell count, 2; mastic negative; colloidal gold curve, 0001000000; Kahn test, negative.

"(h) *Galactose Tolerance Test:* Only very slight trace of galactose eliminated.

"*Diagnosis.*—Chronic manganese poisoning, well advanced." [5]

It is interesting to note that although this man left his manganese exposure in 1918, he still had rather marked disability when examined twenty years later, especially in the use and control of his legs. While many of the symptoms had improved considerably, this case demonstrates that there are certain permanent organic changes.

## Magnesium

Akin to aluminum, magnesium is an extremely light metal which has now become indispensable to many industries, and especially to the aircraft industry. Like cadmium, it is widely used as a protective covering against corrosion.

**Occupational Exposure.**—Workers whose occupation exposes them to magnesium are alloy workers, brick workers, fertilizer workers, glass mixers, paper and fireproof makers, and those engaged in making musical instruments, optical mirrors, explosives and, as aforementioned, those engaged in the manufacturing of airplanes.

**Pathology.**—As far as its systemic effect is concerned, magnesium is a relatively inert metal, or at least to this date no noxious qualities have been demonstrated. It does, however, exert a most peculiar local disturbance when small particles of this substance enter the skin or subcutaneous tissue. According to McCord and associates [10] many metals possess the capacity to liberate hydrogen from water or other hydrous fluids. Following the investigations of foreign investigators prior to World War II, McCord and associates performed excellent experimental studies, adding considerably to the previously existing knowledge of the action of magnesium within the tissues. In their article they quote Bufe and Gissel* as follows:

> "Injuries caused by light metals tend to pursue a course characteristically different from and more severe than those resulting from the heavy metals. The examination of 5,000 instances of such injury has enabled the investigator to establish the fact that two well defined types exist. In the first, a visible particle of metal is found. If the injury be not treated there ensues in two or three days a rapid and progressive inflammation at the site and in the lymphatic system. Vacuole formation occurs as the result of hydrogen formation consequent upon chemical reactions between the metal and the fluids in the tissue. In the second type of injury the causative agent is metal dust which enters a wound resulting

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* Bufe, W. L., and Gissel, H. H.: Injuries Caused by Light Metals; published in Light Metals, 4: 106, 1941.

> probably from some other accident. The wound heals rapidly but a painful granulomatous infiltration which may persist for months occurs. Subsequently these suddenly develop acute symptoms of inflammation as in the first type of injury. Vacuole formation, however, does not occur. Instead granulomatous cells and eosinophiles together at times with giant cells are found."

In McCord and associates' experimental studies, four white adult rats were used. Fine magnesium powder was introduced by a needle into the abdominal wall. Within twenty-four hours, sizeable single or multiple gaseous tumors were observed. The autopsied animals revealed the following:

> "On the ventral surface of each animal there is an irregular elevation measuring between 4 and 7 cm. in diameter and between 1 and 2 cm. in elevation. Each area is crepitant and there is some overlying bluish discoloration of the skin. In each animal this entire area of the anterior abdominal wall is excised. In two instances there was considerable extension of the emphysema into the groins and some gas escaped during excision causing some collapse of the air cysts. After thorough fixation in formalin, blocks of tissue approximately 5 mm. in thickness are cut completely across each specimen in an attempt to get the entire cross section of each lesion.
>
> "Upon microscopical examination it is found that the skin and subjacent subcutaneous zone are essentially normal. Throughout the subcutaneous fascia and muscle there is extensive necrosis associated with marked leukocytic response. In the subcutaneous tissue there are multiple emphysematous cysts measuring up to 2 cm. in diameter. The leukocytic infiltration is most dense about these cysts and there is some actual suppuration into an occasional cyst. A small amount of the injected material is present, but has not produced any appreciable foreign body granulomatous reaction.
>
> "*Microscopical Diagnosis.*—Necrosis of muscle and fascia with extensive subcutaneous emphysema associated with leukocytic infiltration and early suppuration.
>
> "Tissue sections made through the entire tumors revealed many large vacuoles constituting multilocular cysts."

Regarding the nature of the gas produced, McCord and associates write:

> "In previous publications it has been assumed or efforts have been made to prove that the gas generated on contact between magnesium and tissue is hydrogen. Theoretically this should be true, and in truth at the initial formation hydrogen may be the gas generated. On the other hand, the quantity of hydrogen on analysis proved not to exceed 5 per cent. Clearly some gaseous exchange will take place in the course of 48 hours so that the aeroform content of these tumors may not be expected to represent hydrogen alone. Further, due to necrosis such other gases as hydrogen sulfide may be expected to appear.
>
> "The determination of the nature of the gas concerned was carried out in a manner now described along with the results obtained. . . . Seven cubic centimeter Keidel vacuum tubes were utilized for the collection of samples. The furnished degree of vacuum was not sufficient and required re-exhaustion of air. The needle representing a portion of the Keidel apparatus was inserted into the largest gas pocket and the pressure valve on the tube released. Immediate tumor deflation occurred. It was necessary to puncture more than one tumor to

obtain 7 cc. samples. In this manner, a total of 35 cc. of an unknown gas was obtained for examination.

"Two 7 cubic centimeter tubes containing the unknown gas were introduced into one side of a 10 centimeter interferometer cell which had previously been evacuated to 10 millimeters. The Keidel tubes were then completely exhausted by filling with mercury. The unknown gases were made carbon dioxide and moisture free by passing them through a packed tube containing anhydrone and ascarite. The unknown gas was then compared with carbon dioxide moisture free air and the percentages of hydrogen calculated according to a method described by Edwards. The interferometer was calibrated through a method also described by Edwards. Using known refractivities for air and hydrogen, 2.2 per cent of hydrogen was found.

"When this gas was forced from the interferometer cell through a needle and attempts made to ignite the outflowing gas, no combustion was noted, which apparently confirms the result obtained, inasmuch as the lower combustible limit for hydrogen is 4.1 per cent.

"The remaining three tubes of gaseous material were analyzed in a reseach Haldane gas apparatus. The total amount of sample was 23.1 cc. and of this sample 1.3 per cent was found to be carbon dioxide and 15.2 per cent oxygen.

"The indications are that carbon dioxide was generated by tissue degeneration as this quantity of carbon dioxide could not have been introduced from any other source, such as diffusion. The oxygen may have been introduced through a process of diffusion, but this is not definitely known at this time. The hydrogen, which should be the chief theoretical gas, is present in such small amounts as to lead to the belief that extensive diffusion has taken place. These findings do not eliminate the possibility that the initially formed gas was hydrogen."

Wilson and Egeberg[11] believed that magnesium particles are at times disseminated by the lymphatics. They saw one man whose initial lesion was on the right thumb but who subsequently developed a similar lesion on the right mid forearm.

**Treatment.**—Using sterile precautions and preparation of the skin of the involved area, the surrounding area is anesthetized by 2 per cent novocain. The burned tissue is scraped away and all dead or necrotic tissue removed. A careful search for all foreign material should be made and such removed. Scraping of the tissue following the excision of the dead or necrotic tissue should be meticulously done. Sulfanilamide powder or ointment should then be applied before the wound is covered.

## Phosphorus

It is not necessary to devote much space to phosphorus poisoning since it has become a comparatively rare disease. At one time yellow phosphorus was used in the manufacture of matches and fireworks. Outbreaks of "phossy jaw" finally led to the passage of laws which placed a high tax on the manufacture of these articles, and forbade their import or export. Today, red phosphorus and a sulfide of phosphorus, $P_4S_3$, have been substituted for the yellow phosphorus. Red phosphorus is considered nontoxic and the few cases of intoxication which have been reported as being due to red phosphorus are suspected to have been the result of the presence of some yellow phosphorus.

In a "strike anywhere" match the head is tipped with a mixture that is chiefly $P_4S_3$, which is readily ignited by the heat of friction. The main portion of the head is a mixture of combustible material, such as sulfur, rosin, or antimony trisulfide, with oxidizing agents such as lead oxide and potassium chlorate, and glue to serve as a binder. The stick is usually coated with paraffin and impregnated with phosphate, which prevents the stick from continuing to glow after the match has been extinguished.

In the safety match the material in the head is sulfur or antimony trisulfide, with a little potassium chlorate or potassium dichromate as an oxidizing agent, powdered glass to increase the friction, and some glue to hold the mass together. This mixture is not combustible enough to be ignited easily, unless struck upon a box coated with red phosphorus.

Acute phosphorus poisoning is caused by ingestion and therefore is not an industrial disease. Chronic poisoning results from a varying length of exposure, usually after one or many years. This intoxication is characterized by involvement of the osseous system, and frequently results in a necrosis of the jaw bone. During World War II, Heimann[12] had the opportunity to study a case which occurred in New York State. The following case history is taken from his report:

"This 40 year old man had been employed for eight years in the conversion department of a phosphorus plant in which yellow phosphorus was converted into red allotrope. During this time he had had periodic medical and dental examinations none of which had shown any deviation from normal. Dental x-rays had been taken three weeks prior to the onset of his illuess and had been considered normal.

"While on vacation from his job he was advised by his private dentist to have certain teeth removed because he had been complaining of joint pains. This was done. He then returned to the plant dentist complaining of pain in his left lower molar region. The plant dentist x-rayed his jaw and found that there was retention of a molar root. This was removed surgically. At this time the x-ray showed no changes of the jaw bone.

"None of these procedures resulted in relief of joint pains and his jaw pain was aggravated. He, therefore, entered the hospital in which a diagnosis of osteomyelitis of the left lower jaw was made, no attempt having been made to relate the condition to his occupation. A portion of the left mandible was removed surgically. The wound drained a good deal following the operation and he finally left the hospital against advice.

"Since the jaw condition continued to give him a great deal of pain, he again sought medical advice and came under the care of a dental surgeon who, recognizing a relation of his occupation to his illness, treated him accordingly. By this time the condition had extended to involve the right side of the mandible. This dentist instituted 'through and through' drainage and otherwise was very conservative in his treatment. Sequestra were removed as they formed and every effort was made to retain the integrity of the involucrum and periosteum. The dental surgeon described the sequestra as foul-smelling, black structures resembling oven clinkers. He also noted that they were removed with difficulty since there was interlocking of necrotic and healthy bone. One year after the onset of the illness the condition became quiescent.

"At the present time the patient wears a prosthetic appliance to replace all the teeth of the lower jaw and to avoid a facial deformity due to the loss of part of the left mandible. In spite of this appliance there is a deviation of his lower jaw to the left.

"*Comment.*—This case points up the following:

1. It was the first recent case to occur at the plant, and for a short time went unrecognized.
2. A radical surgical procedure was performed in which the integrity of the involucrum and periosteum was not maintained. This resulted in a permanent defect of the jaw.
3. The occurrence of joint pains in this case is interesting in view of the fact that rheumatic pains are said to be found frequently in workers exposed to yellow phosphorus."

## Antimony

True cases of antimony poisoning have been reported only rarely. Most workers exposed to antimony are likewise exposed to other metals.

**Occupational Exposure.**—The usual exposure to antimony occurs where the ore is being mined, smelted, or refined; and also in processes where it is combined as an alloy with other metals, especially lead, tin, and copper. These alloys find use in storage-battery grids, pewters, metal bearings, and type metal. In the defense program it is used especially in the manufacturing of munitions.

**Pathology.**—Most of the references regarding the effects of antimony occur in the foreign literature, and a great deal of it is of an experimental nature. A very recent summary of the literature can be found in an excellent paper by Bradley and Fredrick.[13] These two workers quote the report of Schrumpf and Zabel,* whose observation of workers in a type foundry revealed very few cases of lead poisoning, but a number of men with "remarkable facial expression, complaints of nervousness, irritability, sleeplessness, fatigue, dizziness, muscular and neuralgic pains, loss of appetite, nausea, gastrointestinal disturbance, and constipation." Examination of the blood revealed a diminished leucocyte count and a notable eosinophilia. The blood pressure was lowered. Antimony was recovered in the stools.

A colorimetric method for the determination of antimony in biologic materials is presented.[14] The material is digested in sulfuric acid with the addition of nitric acid or of a mixture of nitric and perchloric acids. The diluted digest is treated with a solution containing potassium iodide and ascorbic acid; the color of the potassium iodoantimonite complex is determined in a photoelectric colorimeter. The standard error is about 2 per cent, and the recoveries of added antimony are usually within plus or minus 2 per cent. Quantities as small as 10 micrograms may be estimated, but the error here is somewhat larger. Bismuth gives the same color reaction as antimony. However, if it is present, it may be quantitatively determined on the same digest, and by subtraction the amount of antimony may be estimated.

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* Schrumpf, P., and Zabel, B.: Clinical and Experimental Studies on Antimony Poisoning of Type Setters, Arch. f. exper. Path. u. Pharmakol. 63: 242, 1910.

Animal studies on the toxicity of inhaled antimony trioxide were carried out by Dernehl, Nau, and Sweets[15] who summarized their work as follows:

> "Twenty-four guinea pigs inhaling antimony trioxide in amounts varying from 13 to 424 mgms. showed the following findings: (1) Extensive pneumonitis in all animals. (2) Fatty degeneration of the liver in 11 out of 15 animals having 'exposures' of 77 mgms. and above. (3) Hypertrophy of the lymphoid follicles of the spleen associated with a decrease in the number of polymorphonuclear leukocytes. (4) A decrease in the number of polymorphonuclear leukocytes and eosinophiles in the circulating blood with a relative increase in the number of lymphocytes. (5) A decrease in the total white blood cell count. (6) No pathologic changes demonstrable in the heart or kidneys. (7) Normal electrocardiograms in three exposed guinea pigs. (8) Death of four out of 24 animals—a mortality rate of 16 per cent. (9) Death from pneumonia occurring much more frequently in treated animals than in untreated animals. Electrocardiograms taken on six exposed employees in a smelter showed five out of the six to be normal. The greater toxicity of inhaled dust as compared to ingested dust is discussed."

The degree of pathology indicated by animal experimentation has not been observed clinically.

## Zinc

The acute intoxication in which zinc plays a part has been described under Metal Fume Fever.

**Chronic Poisoning.**—Chronic zinc poisoning is doubted by most investigators. In the older textbooks and literature, instances of chronic zinc poisoning were recorded. It is now felt that other metals present in the process in which zinc is used provide the intoxication. In 1926, McCord and Friedlander[16] studied the employees in two galvanizing plants. In one plant, where the working conditions were poor and the men had been engaged in this work for a long period of time, they exhibited gastric disturbances varying from gastroenteritis to gastric and duodenal ulcers. In the second plant, where the hygienic conditions were fairly good and the men had a short term of exposure (less than six years), no gastrointestinal disease was found. They concluded that a long period of time expressed in years (five to twenty), plus poor working conditions and a poor type of worker, favored the production of chronic zinc poisoning. In that same year, Batchelor[17] and others found no acute or chronic illness attributable to zinc in twenty-four workmen exposed from two to thirty-five years. Writers in the recent literature are of this same opinion, although duBray[18] reported a case in 1938, believed by him to be due to chronic zinc intoxication.

Zinc is found in various human tissues and the excreta. Some zinc is found in practically all food. In industry, zinc is involved in the process of mining, smelting, manufacturing of brass and other alloys, galvanizing, and in many other less important procedures.

Anyone alleging illness due to zinc warrants thorough study for causes not industrial in origin, as well as careful inquiry into the process to which he is exposed in order to determine the presence of other metals.

**Medicolegal Aspects.**—An allowable concentration is 14 mg. per cubic meter. Temporary disability is short, a few days at the most. There is no permanent disability.

**Treatment.**—

PROPHYLAXIS.—The preventive measures are the same as those to be adopted with a lead hazard, or any dust hazard likely to contain lead. Adequate ventilation, avoidance of the deposits of dust, and frequent cleaning of all parts of machinery, floors, and walls of dust collections are factors in reducing exposure. Men should be selected who are free from focal infection. Some authors advise periodic changes of work and comparatively short working hours. The inclusion of adequate fat and milk in the diet is thought to prevent to some degree the occurrence of symptoms in those working with zinc.

If a case of *chronic zinc poisoning* has been established, the patient should be removed from his hazard at once. Anemia, if present, is treated by ferrous sulfate, 5 grains (0.32 Gm.), two tablets three times a day. In *acute intoxication*, a few days' absence from the work and treatment as described under Metal Fume Fever will suffice.

**Zinc Chloride Burns.**—Treatment of zinc chloride burns consists of removal of the necrotic slough and of filling the wound with sodium bicarbonate. Compresses of a warm solution of sodium bicarbonate are then used. After the lesion has been cleaned up to some extent, it is dressed with borated petrolatum, painted with merthiolate, or covered with metaphen in collodion. Gloves should be worn by workers handling flux containing zinc chloride. When zinc chloride powder is handled, ordinary washing with soap and water will not remove it as a rule, while a 5 per cent solution of hydrochloric acid will.

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# CHAPTER XXV

# MERCURY, CHROMIUM, COPPER, AND ALUMINUM

## Mercury

Mercurialism is one of the oldest of all occupational diseases. Paracelsus described it in 1567, but the best of the early descriptions was given by Ramazzini. The first episode of mercurialism in America occurred among the fur cutters and hatters in 1859.

**Occupational Exposure.**—There are about 100 occupations in which workers are exposed to this metal. All of these need not be listed. Table XV lists but a few of the hazards and shows the form of mercury used.

TABLE XV

| | |
|---|---|
| Mercury | Manufacture of scientific instruments |
| Mercury nitrate ($HgNO_3$, $Hg(NO_3)_2$) | Felt hat industry; decorating porcelain |
| Mercury fulminate ($Hg(CNO)_2$) | Constituent of all percussion caps |
| Mercury sulfide (Cinnabar, $HgS$) | Mining and used as a pigment |
| Mercury cyanide ($Hg(CN)_2$) | Making cyanogen |
| Mercury chloride ($HgCl_2$) | Manufacturing of antiseptic dressings |
| Hatting industry ($Hg(NO_3)_2$) | Treatment of felt |
| Fire gilding | Applying gold mercury amalgam to base metal and driving off mercury by heat |
| Mercury amalgam | For extracting gold and silver from ore |
| Mercury pumps | To exhaust air from incandescent lamps |
| Chemical works (solder for batteries) | As $HgCl_2$, etc. |
| Dentists, mercury boilers, welders | Mercury amalgams |

**Pathology.**—Subacute or chronic mercury poisoning usually affects the mucosa of the mouth and also the nervous system. Severe gastrointestinal inflammation and irritation to the kidneys are not found in industrial poisoning; these signs follow the ingestion of mercury which is a rare incidence in industry. No characteristic disturbances of the blood cells are noted in industrial exposure.

**Signs and Symptoms.**—Stomatitis has long been considered as a classical sign of mercurialism together with excessive salivation. Although Neal and Jones[1] did not find this to be true in a series of workers in the hatters' fur-cutting industry, the signs and symptoms indicating involvement of the nervous system are by far the commonest, among which tremor is the most startling. This tremor becomes intensified by attention or intention. This is an early sign of mercury poisoning and its presence can be demonstrated by having the workman attempt to write or draw straight or curved lines.

The psychic disturbances include restlessness, irritableness, fear, lack of concentration, lack of confidence, and depressive states. In the physical examinations of 529 fur cutters, Neal and Jones found the incidence of nervous system involvement (percentage) to be as shown in Table XVI.

TABLE XVI

| | | | | |
|---|---|---|---|---|
| Fine intention tremor | | | | |
| Of any part of the body | 100.0 | ... | 43 | 0 |
| Of fingers | 79.1 | ... | 34 | 0 |
| Of eyelids | 62.8 | ... | 27 | 0 |
| Of tongue | 46.5 | 0.0 | 20 | 0 |
| Of other members | 25.6 | ... | 11 | 0 |
| As shown by shaky signature | 37.2 | 5.5 | 16 | 22 |
| Psychic irritability | 79.1 | 1.0 | 34 | 5 |
| Speech defect | 18.6 | 0.4 | 8 | 2 |
| Exaggerated knee jerk | 20.9 | 4.3 | 9 | 21 |
| Positive Romberg test | 2.3 | 0.4 | 1 | 2 |
| Dermatographia | 11.6 | 0.8 | 5 | 4 |
| Abnormal blushing | 25.6 | 0.2 | 11 | 1 |
| Excessive perspiration | 11.6 | 0.2 | 5 | 1 |

**Laboratory Tests.**—

MICRODETERMINATION OF MERCURY IN BIOLOGICAL MATERIAL.—

"Organic matter is destroyed by digesting the sample in a special apparatus, by boiling first with a mixture of sulfuric and nitric acids, followed by a second boiling treatment after addition of potassium permanganate. Mercury is extracted from the decolorized acid-permanganate digest (diluted to 1.8 N in respect to sulfuric acid) by means of a chloroform solution of di-beta-napthylthiocarbazone. The chloroform extract is then treated with a sulfuric acid-sodium thiosulfate mixture to separate the mercury from copper. The final estimation of mercury is made by photometering di-beta-naphthylthiocarbazone-chloroform extracts of the copper-free aqueous phase following treatment with potassium permanganate. At least 50 Gm. of blood must be used for the reliable estimation of concentrations as low as 1 microgram of mercury in 100 Gm. of blood. A table, which compares the mercury content of the blood and urine of exposed persons, shows that the mercury content of the blood is very low. Except in one case, the amounts were well below 10 micrograms for 100 Gm. of blood. The highest, 0.029 mg. per 100 Gm., was found in a case where the urine level was 4.44 mg. of mercury per liter. The mercury-di-beta-naphthylthiocarbazone complex is more stable than the analogous dithizone complex."[2]

DETERMINATION OF MERCURY IN URINE.—"To 500 cc. of urine add 50 cc. of 30 per cent sodium hydroxide, 10 Gm. of crystalline sodium hyposulfite, 2 cc. of 10 per cent sodium sulfate, and 1 cc. of 20 per cent barium chloride solution. Heat on a boiling water bath 1½ hours, shaking occasionally, add two or three more drops of barium chloride solution, and let it stand over night to cool and settle. Decant, collect, and wash the precipitate of barium sulfate with its entrained mercury in a centrifuge tube, and dry the precipitate in the tube at room temperature by a stream of air. To the dry precipitate add 2-3 drops of pure promine to convert the mercury to mercuric bromide, evaporate the excess bromine, extract the mercuric bromide with ether, evaporate the ether, and determine the mercury in the evaporation residue by nephelometric or other known methods."[3]

**Treatment.**—No treatment for mercury poisoning has ever been specific, but in 1946 Loncope and Luetscher[4] demonstrated the effectiveness of BAL in the treatment of mercury poisoning. The cases treated were all of the acute type. The actual value which BAL will finally prove to have in industrial (chronic) poisoning is not yet known. For the dosage of BAL the reader is referred to the chapter on arsenic. The following general regime is advocated: Removal from the hazard is, of course, absolutely essential. The use of sodium thiosulfate might be of value, 15 grains (1 Gm.) in 10 cc. of sterile water intravenously every other day for a short time.

NERVOUS DISORDERS.—Most of the treatment in the chronic case is directed toward alleviation of the nervous affections. Since many of these patients have symptoms similar to those seen with chronic encephalitic parkinsonism, it has been suggested that a trial of drugs used in that disease be made. The older drugs of that group are scopolamine hydrobromide (hyoscine), from 1/200 to 1/100 grain (0.0003 to 0.0006 Gm.), two or three times per day, or tincture of stramonium, from 60 to 90 minims, three times daily. A drug used more recently has been amphetamine sulfate (benzedrine sulfate), from 20 to 30 mg. twice daily, at 8 A.M. and at noon, in conjunction with a drug of the atropine group, or genoscopolamine, in dosage of 0.5 mg. granules, one three times a day, increasing the dosage after several days to two or three granules three times a day. The latter is said to have far less toxic effects than atropine, hyoscine, and stramonium.

### Case Histories.—

CASE 1.—In 1941 a 57-year-old male reported to the author with almost complete loss of the nails of the fingers and toes, slight exfoliation of the skin of the feet, and the loss of all teeth. The teeth had been removed by a dentist four months previously because they were "loose and had pyorrhea."

The company for which this claimant worked is engaged in a process of obtaining mercury and antimony by the reduction of livingstoneite concentrate, obtained from Mexico. This workman would take sacks of concentrate to a scale to be weighed and then to the elevator where the material was carried to a platform where it was dumped into a hopper. He then carried the cinders which fell to the bottom of the furnace to a pile where they cooled and were re-sacked. These cinders when hot gave off a strong sulfur fume. The cinders also contained antimony that had been separated from the mercury. For ten minutes every hour the patient worked at the hoe table where mud containing mercury was spread out and under which was a fire. Also, every hour he drained mercury from the condensers into a flask.

The physical examination of this workman revealed no abnormalities other than those mentioned. Likewise, the blood and urine studies of this patient were entirely normal.

In the absence of any evidence of involvement of the nervous system it was difficult to ascribe this patient's disturbance to the finger nails to the action of mercury. Dr. Nelson Paul Anderson, a Los Angeles dermatologist who saw this patient in consultation, stated that onychomadesis had never been ascribed to mercury poisoning but that in this case it might be due to antimony or arsenic, both of which the patient handled in the course of his work. The patient was subsequently presented before a joint meeting of the Los Angeles and San Francisco Dermatologic Societies,[5] at which time Dr. M. Maynard voiced the consensus of opinion by stating that the condition was due to the absorption of several metals, mercury, arsenic, and antimony, rather than to be a contact dermatitis.

It is interesting to note that in discussing the paper of Neal and Jones,[1] previously referred to, McCord pointed out that in the cartridge industry the mercury fulminate is in a wet state, and therefore the type of exposure is somewhat different from what it is where fumes are in existence. He stated that in those who handle the wet mercury fulminate, ulcerated fingertips, erosion of the nails, and sore fingers appear.

In 1945, Lewis[6] made a study of mercury poisoning in the tungsten-molybdenum rod and wire manufacturing industry, and the following case report is taken from that study.

CASE 2.—"*Chronic Mercurialism.* A. H., a white man aged 59, on January 27, 1943, was hospitalized because of weakness, tremor and painful gums. He was employed in the treating department for approximately eight years and worked continuously until January 23, 1943. The plant where he was employed was ventilated only by means of windows situated behind a row of treating bottles, and during the winter the windows were opened only in mild weather. Early in 1942 the patient first noted soreness and dryness of the throat, and somewhat later he began to complain of periodic gastric distress. Ten months prior to examination he first became aware of tremor. His family physician was unable to find any physical abnormalities. Soreness of the mouth became progressively worse, but the diagnosis remained obscure. The past medical and family history were non-contributory. There was no history of alcoholism. The systemic review was negative.

"On February 10, 1943, physical examination revealed that the patient was well muscled, weighed 145 pounds (66 kg.) and was 66 inches (168 cm.) tall. The blood pressure was 114 systolic and 76 diastolic. The temperature and pulse were normal. Both eyes showed immature cataracts. Hearing was slightly impaired. There were a few missing teeth. Dental occlusion was poor. There was moderate recession of the lower anterior gums and a faint gray line along the margin of the receded tissue. The gums did not bleed easily on traumatization. The tonsils were of moderate size, not inflamed. The lung fields were clear. The cardiac examination was negative. The liver margin was palpable 1 fingerbreadth below the costal margin. The abdomen was otherwise normal. The genitalia and extremities were normal. The peripheral arteries were not unduly thickened.

"The patient was of good intelligence and there were no psychiatric abnormalities except tension and anxiety. There was no disturbance of gait or station. There was a very noticeable intention tremor in the finger-to-nose test, and there were coarse tremors of the outstretched hands of intention type and of variable amplitude. During the course of observation and on attempted performances of purposive acts the tremor became much more pronounced and spread to involve almost all the muscles of the neck and trunk. There was no motor weakness. The cranial nerves were normal. The deep reflexes of the upper extremities were hypoactive, while those of the lower extremities were somewhat hyperactive. There was abortive clonus at the achilles and patellar tendon. There was an equivocal Babinski reflex on the left and a positive Babinski reflex on the right.

"The laboratory findings were essentially negative. There was no anemia. The leukocytes numbered 13,400 per cubic millimeter and there was a slight shift to the left. The sedimentation rate was 7 mm. per hour. The Kahn and Hinton reactions were negative. The blood sugar was 106 mg. per hundred cubic centimeters. Urinalysis revealed a specific gravity of 1.014, no albumin or sugar, and an occasional hyaline and coarse granular cast. Examination of the urine for mercury by the dithizone method gave a negative reaction. (At the time of examination the patient was hospitalized and had not been exposed to mercury for several weeks.)

"The patient was re-examined on March 18, 1943, at which time he presented a similar but somewhat less pronounced tremor. There was no change in the physical findings. The handwriting at that time showed pronounced tremor, and the signature was illegible. In the drawing of straight and curved lines there was progressive and decided irregularity."

## Chromium

Chromium is found in several materials, chiefly as chromic iron or chromite. Industrially the bichromates of potassium and sodium and chromic acid concern us. Chromium as a metal is inert, but chromic acid and its compounds, because of their caustic action, affect the skin and mucous membranes.

**Occupational Hazards.**—In the manufacturing of coal-tar dyes, wallpaper, electric batteries, and explosives; in textile printing; in certain paints, such as lead chromate; in lithography; and, most commonly of all, in photography and electroplating.

**Signs and Symptoms.**—There have been a few cases reported in the literature where some form of the chromium has been ingested with serious or fatal results. These do not deserve consideration in this discussion.

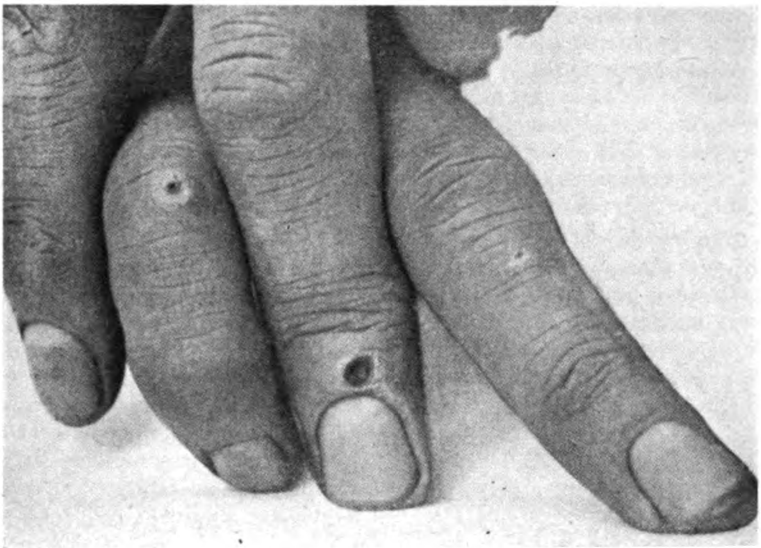

Fig. 36.—Healing chrome holes. The permanent scarring is well seen. (White: The Dermatergoses or the Occupational Affections of the Skin, New York, 1938, Paul B. Hoeber.)

During World War II, Zvaifler,[7] as consulting otolaryngologist to the Elastic Stop Nut Corporation, examined over 100 men exposed to chromic acid and having incurred either a chromic ulcer or a chromic skin rash, or both. He found no instance of constitutional disturbance.

ULCERS.—Industrially, the acid produces lesions around the base of the nails of the hands, on the knuckles, on dorsum of the hands, and on the forearms. Occasionally the ulcers affect the eyelids and often the nasal mucosa. They usually develop slowly, and the workman is often unaware of their presence until they become big enough to note upon inspection (Fig. 36). Sometimes they may be painful, especially if they occur over the knuckles, where flexion is hindered. These ulcers may become fairly deep. The ulcers are usually small, varying in size from the head of a match to the end of a lead pencil, but often quite deep. The edges are thick, rounded, often glossy and rarely inflamed. They may persist indefinitely unless treated. On healing there is usually a permanent residual scar. Invariably they cause no apprehension until they occur in the cartilaginous portion of the nose.

**Medicolegal Aspects.**—

TEMPORARY DISABILITY.—This is short. Treatment usually stops the progress of the lesions, and removal from exposure or the employment of preventive measures permits the affected person to continue his work.

**Treatment.**—

SCRUBBING TISSUE.—If the lesion or lesions ("chrome holes" or ulcers) are small and superficial, the damaged tissue may be scrubbed thoroughly with 5 per cent sodium hyposulfite, water, or saline solutions. The purpose of this treatment is to dislodge all tissue containing chromium compounds so that additional damage by burrowing will not occur. After this scrubbing, the wound may be treated like any abrasion of similar severity.

WET DRESSINGS.—If the ulcers are already too deep for such scrubbing, wet dressings of sodium hyposulfite for a period of from three to five days should be applied to aid in the reduction of the chromium compound. Since the chromium in the flesh is not dislodged by sodium hyposulfite, wet dressings of from 5 to 10 per cent solution of sodium citrate, sodium lactate, or potassium and sodium tartrate should be applied to accomplish this. These wet dressings are continued for from three to five days. At times necrotic tissue in the ulcer will require curettage. Suppuration rarely occurs in chrome ulcers, but cleansing with a mild antiseptic agent following use of the reducing agents may be used. Treatment appropriate for any ulcer is used following the therapy outlined. Vitamin A and D ointment is beneficial.

It is frequently desirable to remove the worker from his customary duties, at least during the early stages of treatment.

PROPHYLAXIS.—Prophylactic measures consist of removal of fumes and dust from the atmosphere, of cleanliness within the plant, and of confining the process within enclosures wherever possible. Respirators or gasmasks should be worn. Shower baths and change of clothing should follow the day's work, and the installation of showerbaths and locker rooms will encourage this procedure. The skin should be protected by suitable clothing, rubber gloves, and protective ointments or oils. The nasal mucosa should be sprayed with an oil prior to going to work.

## Copper

Copper is commonly used in industry in various forms. Bronze is a copper alloy containing tin and sometimes zinc; brass contains copper and zinc. There is no evidence that copper, as used in industry, causes any systemic reaction. Illness attributed to copper is invariably due to arsenic, lead, or other substances associated with copper in the industrial process. This statement likewise applies to many skin disorders attributed to copper.

Inhalation of cuprous oxide has been claimed as a source of irritation to the upper respiratory tract as well as of conjunctivitis. Discoloration of the skin in those handling copper is often seen but this does not indicate any actual injury from copper.

The effect upon the liver as a result of prolonged feeding of copper salts has been studied experimentally with the conclusion that a pigment cirrhosis resembling closely the early manifestations of hemochromatosis in man will result. Again, it must be stated that there exists no industrial poisoning similar to these experimental findings.

## Aluminum

As a light metal, aluminum is inert and causes no harm. It has been used as a dust in the attempts to prevent or minimize the effects of silica in the lungs. (See the chapter on Silicosis.)

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# CHAPTER XXVI

# SELENIUM, TELLURIUM, VANADIUM, AND RADIUM

## Selenium

Selenium has received scant attention from industrial toxicologists. On the other hand, since it is a natural constituent of the soil of many of the western states of the United States, its effect upon the rural population has been studied rather extensively. These effects are now well recognized. Dudley[1] stated that in the processing of selenium-bearing products, there exists an unrecognized hazard to workers.

**Occupational Exposure.**—Following are some of the industries in which workers are exposed to selenium: Glass decolorizing, production of ruby glass production of red and yellow glazes, production of paint and ink pigments, production and coloring of plastics, manufacture of photoelectric apparatus, alloying of machineable stainless steels, alloying of free machining copper base alloys, fireproofing of electric cable.

**Pathology.**—The pathology of selenium has not, as yet, been adequately studied, although considerable animal experimentation has been done. There is, apparently, a divergence between its action upon animals and that upon human beings. In animals, subacute poisoning has produced bronchial pneumonia, fatty degeneration of the liver, degeneration of the kidney cells, and other minor alterations, which clear up without residual pathology. Gastrointestinal disturbances noted in the human being have been absent in animal experimentation.

**Signs and Symptoms.**—These are respiratory irritation, cough, edema of the lungs, vomiting, diarrhea, abdominal pain or cramps, loss of reflexes, cerebral irritation as manifested by convulsions, and death.

ACUTE REACTIONS.—Concerning the severe reactions resulting in convulsions or death, one finds these mentioned, but the author was unable to find any reference to actual death from selenium poisoning other than that of Gadamer in 1909. The garlic odor to the breath has been frequently referred to.

CHRONIC REACTIONS.—Possibly the best picture of the chronic effects from constant exposure is reported by Smith, Franke, and Westfall.[2]

"A series of 111 families was studied for clinical evidence of selenium intoxication, and a series of 127 specimens of urine of as many subjects, representing ninety families, was analyzed for this element. The results of the urinary analysis showed that only 8 per cent of the cases were free, or nearly free, of selenium, while 92 per cent contained amounts varying from 2 to 133 micrograms of selenium per 100 cubic centimeters. This affords definite proof of the absorption of selenium by some of the rural population. . . . In the 111 families visited, the following disorders, exclusive of the more vague symptoms of anorexia, indigestion, general pallor, and malnutrition, were observed:

"1. Bad teeth, varying from marked discoloration through all stages of decay, were seen in one or more members of forty-eight families.

"2. Yellowish discoloration of the skin, in many cases very definite icterus, and in some cases seemingly associated with more or less definite liver disease, was seen in about forty-six subjects.

"3. Skin eruptions of varying degrees of severity, but not conforming to any one particular type, were seen in twenty subjects.

"4. Chronic arthritis with more or less permanent changes in the joints was present in fifteen subjects, varying from the milder types of rheumatoid arthritis to a more severely deforming type of arthritis deformans.

"5. Diseased nails of the fingers and in some cases also of the toes, were observed in eight subjects. They were usually asymmetrical, atrophic, brittle, and irregular, and often presented transverse and, at times, longitudinal ridging.

"6. Subcutaneous edema of probably cardiorenal origin was diagnosed in five cases and peripheral neuritis of doubtful etiology in two subjects. Fifteen subjects gave a history of more or less protracted gastrointestinal disturbances."

While this study of Smith, Franke, and Westfall[2] cannot be considered an observation of industrial exposure, yet it does offer the largest group of cases yet noted in the literature, and many of the symptoms are analogous to those resulting from industrial exposure. Certain observers have noted a close similarity between selenium and tellurium poisoning. Alice Hamilton,[3] in 1917, investigated selenium poisoning in copper foundries. She reports the chief symptoms were bronchial irritation and digestive disturbances.

K. Halter[4] reports the case of a man working as a mixer in a plant where colored glass was produced. After working with elementary selenium and especially with sodium selenite, used for bleaching greenish glass, he developed headaches, then irritation of the mucosa. After about two days, reddening and swelling of the uncovered skin followed. These symptoms appeared often and increased in severity.

Clinical examination disclosed edematous erythema of the face and neck, and several hard, infiltrated, ill-defined foci on the back of the hands and fingers. No change appeared on skin protected by clothing. Nasal and laryngeal mucosa was reddened and somewhat thickened. Slight conjunctivitis was present. There was an enlarged liver and increase of porphyrine in the urine. Selenium was detectable in the urine. Basal metabolism was increased. There were no nervous or gastrointestinal symptoms (in contrast to arsenic poisoning). Inflammation of nasal mucosa and conjunctiva is the direct result of the irritating action of selenium, as is the injury to liver function.

Regarding the absorption of selenium, Sterner and Lidfeldt[6] have sounded an important warning:

> "During a routine survey of potentially hazardous chemical exposures, selenium was found in the urine of a chemist who was synthesizing an organo-selenium compound. Chemists snythesizing methylbenzoselenazole absorbed and excreted in the urine considerable amounts of selenium. The absorption occurs almost entirely through the intact skin. Protective devices such as rubber and synthetic rubber gloves give only temporary protection, as the compound readily penetrates them. By requiring frequent changes of new gloves, protective cream and scrupulous care in handling, the absorption has been materially lessened, but complete prevention of such absorption and excretion is not yet accomplished. There have been no symptoms or signs suggestive of toxic effects in any of the workmen so exposed, nor have serial comprehensive blood studies or hepatic and renal function tests indicated any injury. In one case the urinary excretion averaged 5 mg. of selenium a day for a period of a week; I believe this is a greater amount than has been reported previously in the human being. To demonstrate the skin absorption I dropped 0.1 cc. of the selenazole (the vapor pressure is negligible) containing about 50 mg. of selenium on my hands, removing it by thorough scrubbing after thirty minutes. During the next four days over 5 mg. of selenium was excreted in the urine, with trace amounts only in the stools."

*Tests.*—It was believed that the finding of selenium in the urine indicated "conclusive evidence of the absorption of selenium by workers" (Dudley[1]). But according to Sterner and Lidfeldt[6] "selenium was found in a high percentage of urine specimens obtained from normal, healthy male adults who had incurred no industrial or otherwise obvious selenium exposure. This finding is in contrast to recent references on the subject. The concentrations of selenium averaged from 4 to 5 gamma per 100 cc. of urine, with a range of 0 to 15 gamma. The rate of urinary selenium excretion is relatively constant and is quite independent of the urinary volume, suggesting that selenium is treated by the kidney as a 'nonthreshold' substance similar to sulfates and creatinine. The mean rate was 2.2 gamma per hour with relatively slight variability. A dietary source of selenium was found in wheat products, with selenium values ranging from 26 to 39 gamma per 100 Gm. dry weight or 0.26 to 0.39 p.p.m. No harmful effects were observed which could be correlated with the absorption and excretion of such small amounts of selenium as were demonstrated in the investigation."

**Treatment.**—

PROPHYLAXIS.—The air breathed by the workers should be free of this substance, or its presence reduced to a minimum. Frequent sampling is necessary to accomplish this. If this substance is subjected to high temperatures, exhaust ventilation should be in operation. The skin should be protected to prevent involvement as noted by Halter.[4] Workmen with known respiratory conditions, impaired liver function, or arthritic tendencies should be excluded from this hazard.

In chemists synthesizing organoselenium compounds, absorption through the skin is prevented by frequent changes of new gloves, protective cream, and scrupulous care in handling products. Protective devices, such as rubber and synthetic rubber gloves, give only temporary protection, since some compounds, for example, methylbenzoselenazole, readily penetrate them.

DIURETICS AND CATHARTICS.—No specific treatment for this poisoning is outlined, but if intoxication has occurred, removal from exposure with possible hospitalization is necessary. The use of diuretics to secure adequate elimination may be indicated; for example, ammonium chloride, 15 grains (1 Gm.), from four to six times daily, or theophylline, 3 grains (0.195 Gm.), three times daily. In this regard saline cathartics are useful, such as magnesium sulfate, from ½ to 1 ounce (16 to 32 Gm.); the effervescent preparation of magnesium citrate, 12 ounces; sodium phosphate, 1 drachm (4 Gm.); or the effervescent preparation of sodium phosphate, 2½ drachms (10 Gm.).

RELIEF OF SYMPTOMS.—Upper respiratory symptoms may be treated as described under Cadmium. Intravenous 50 per cent glucose (50 to 100 cc.) may aid in treatment of edema of the lungs, as may from ½ to 2 cc. of salyrgan intravenously at from three- to four-day intervals. If kidney degeneration is present, the use of salyrgan is probably contraindicated, although some believe that salyrgan may be used even in the presence of definite kidney pathology. Inhalations of oxygen by intranasal tube or by a tent may be needed, and intravenous infusions of 10 per cent dextrose in physiologic saline (1,000 to 3,000 cc. daily) may be necessary if vomiting or diarrhea is present.

DIET.—The use of a high carbohydrate diet with an increased water intake has been suggested, as has been the use of concentrates of the vitamin B complex. Experimental work[7,8] on animals indicated the protective action of a diet high in protein and low in carbohydrate against selenium. There was also some evidence that the ratio of selenium to the protein in the diet seems to determine the toxicity of of selenium in food and that the quality of protein, as well as the quantity, was of importance. For example, casein and lactalbumin tend to counteract the toxicity of selenium while edestin and gelatin are not effective.

The trial of similar diets in selenium poisoning in human beings has not been reported, but the use of diets high in protein, such as casein, lactalbumin, etc., might well be worth while in treatment of these patients. In addition, when liver damage is present, the use of a high carbohydrate, high protein diet should be followed. Also experimentally, sodium arsenite, five parts arsenic per million of drinking water, completely prevented symptoms of selenium poisoning. Clinical use of this has not been tried.

### Tellurium

Tellurium is metallic in its appearance but in chemical behavior it occupies an intermediate position between the metals and nonmetals. It is a member of the sulfur family, as is selenium.

**Occupational Exposure.**—Tellurium may be used by copper refiners, glass colorers, lead refiners, rubber makers, zinc refiners, and photographic workers. More recently it has been employed in the production of agricultural implements, iron castings, car wheels, and certain types of steel.

**Pathology.**—Very little study of the effect of tellurium upon the human organs has been reported. The most comprehensive report which has appeared in the American literature is that of Steinberg and associates.[10] They cite animal experimentations carried out by Mead and Gies who found that:

> "Tellurium compounds did not affect the metabolism of dogs brought to a state of nitrogenous equilibrium, even when the dosage of orally administered tellurious oxide over a period of a week reached a total dosage of 1.05 Gms.*
>
> "Larger doses by mouth retarded gastric digestion, induced violent vomiting, loss of appetite, somnolence, and produced odor of the breath and sweat.*
>
> "Large doses, 1.45 Gm. injected subcutaneously in dogs in a period of 4 hours, produced over a three-day interval symptoms of restlessness, tremor, diminished reflexes, paralysis, convulsions, somnolence, unconsciousness, cessation of respiration, and death from asphyxia. This is the picture of acute tellurium poisoning in animals.*
>
> "The pathologic lesions observed have been a hemorrhagic gastroenteritis, with dilatation of the capillaries of the splanchnic area, disintegration of the mucous membranes of the gastrointestinal tract and pigmentation of the lining mucosa and intestinal contents and excrements with metallic tellurium.* Certain investigators have reported that in heavily exposed animals terminally there is a severe destruction of red blood cells and diminution of the hemoglobin.
>
> "The pathologic findings have presented themselves in clinical symptoms in animals as loss of appetite, vomiting, diarrhea, and constipation."

According to Steinberg and co-workers[10]:

> "Tellurium and its compounds may enter the body by inhalation and then directly into the systemic circulation, by ingestion, and through the skin. Glandular and tissue cells are able to reduce soluble tellurium compounds coming in contact with them to metallic tellurium. Animal experimentation has shown that these cells also play a role in the formation of methyl telluride, although this function is believed to be principally performed by the liver. Experimentally, toxic effects of tellurium have been observed during the process of the reduction of the tellurium compounds to the metal. However, during the period of conveyance of

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[*] Mead, L. D., and Gies, W. J.: Physiological and Toxicological Effects of Tellurium Compounds, Am. J. Physiol. 5: 104, 1901.

stored tellurium and its introduction into the blood stream as circulating tellurium, and subsequent excretion via the urine, feces, breath, or sweat—no matter in what form the compound, it may exert toxic effects.

"Tellurium is readily soluble in the tissue fluids and may be carried to, and deposited in, all organs. Experimentally it has been shown that after subcutaneous injection, tellurium may be recovered from the muscles and skin at the point of injection, from the liver and kidneys, stomach, and brain tissue. It is present in the blood, urine, and bile.*

"In the breath and sweat it is excreted in the form of methyl telluride with the characteristic odor present."

Sixty-two men were examined by Steinberg. These men were subjected to varying degrees of exposure to tellurium. The portal of entry into the body was the respiratory tract. The subjective symptoms in this group were "garlic odor of the breath, dryness of the mouth, metallic taste, somnolence, garlic odor of the sweat, loss of appetite, and nausea. The first five were encountered frequently, and are considered as symptoms of tellurium absorption occurring at concentration ranges encountered in the workroom air in this investigation. Loss of appetite was not a constant symptom, occurring in five individuals who were only lightly exposed, and for which other illnesses might have been responsible. In the four heavier exposures, loss of appetite was not complained of at the time of the second examination. The nausea complained of in two workers, who were close to the source of fume emanation, was transient in character, and had occurred three or four times within the past year of exposure. It was not associated with abdominal pain, vomiting, or diarrhea. Constipation was not a subjective complaint in any of the ninety-eight workers exposed to various concentrations of tellurium.

### Vanadium

There is not much American literature regarding the effects from exposure to vanadium dust. Dutton[11] reported a case in 1911. He, with others, has indicated an irritating cough, gastrointestinal symptoms, and anemia to be present following exposure. In contrast to these reports, the investigation of Symanski[12] suggests that the affection is confined to the eye, the upper respiratory tract, and the lungs.

**Vanadium Pentoxide as Toxic Product.**—After calling attention to the fact that the use of vanadium has greatly increased in recent years, Symanski[12] reviews the literature on the disorders supposedly caused by it. Reviewing the complicated procedure by which vanadium is produced, he shows that of the various transformation products, it is only the vanadium pentoxide, particularly in the readily absorbable form of dust, which is harmful to the health of the workers. He subjected the workers who came in contact with this substance to a careful examination. He describes observations on nineteen men.

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* Mead, L. D., and Gies, W. J.: Physiological and Toxicological Effects of Tellurium Compounds, Am. J. Physiol. 5: 104, 1901.

**Signs and Symptoms.**—Symanski[12] found a number of characteristic disorders. He observed conjunctivitis with inflammatory injection of the conjunctivae, suppurating discharge, and corresponding complaints such as burning of the eyes, rhinitis with reddishness of the nasal mucosa, and coryza with watery discharge; also a feeling of soreness in the pharynx, continuous coughing with more or less profuse expectoration, occasionally sanguinous pharyngeal secretion, a feeling of dryness in the pharynx, sensations of constriction of the chest, occasionally piercing pains, the typical aspects of a more or less severe subacute or chronic bronchitis, considerable sonorous and sibilant râles in the absence of signs, indicating inflammatory infiltration of the pulmonary parenchyma. He observed no tuberculous complications, but there were roentgenologic signs indicating a chronic bronchitis.

In view of the fact that all examined persons had been active in the production of vanadium for only a few years, Symanski[12] thinks that it is necessary to consider the possibility of the development of chronic changes. Contrary to reports in the literature, he never observed reabsorptive gastrointestinal disturbances, renal symptoms, increased nervous sensitivity, disturbances of the central nervous system, or impaired vision. Moreover, he found no characteristic changes in the composition of the white or red blood picture, especially signs of anemia.

According to Wyers[13] the features of the syndrome associated with industrial exposure to vanadium pentoxide are: (1) pallor of the skin; (2) greenish-black discoloration of the tongue; (3) paroxysmal cough, rarely with hemoptysis; (4) dyspnea; (5) pains in the chest; (6) palpitation on exertion; (7) tremor of the fingers and arms; (8) barrel-shaped emphysematous chest; (9) profuse bronchitis; (10) bronchospasm; (11) raised blood pressure, accentuated pulmonary sound, and (12) reticulation on x-ray examination. It seems likely that colds and pneumonia are of more frequent occurrence than in the general population.

## Radium

Radium is found in nature in association with uranium.[14] Deposits in the Congo State and in Central Africa and deposits discovered in 1931 on Hudson Bay, in Canada, are the main source of the element. The chief ore is carnotite, an impure potassium urano-vanadate which occurs as a cementing material in sandstones.

In the past, such low-grade ore was used that about 250 tons were required for each gram of radium. The price of radium has recently been materially reduced by the opening of new deposits. In 1940, the price was $30,000 for one gram of the element. This means that a quantity of pure radium equal in weight to a postage stamp may be purchased for about $1500. In practice, radium salts are always put on the market in admixture with the corresponding salts of barium.

When radium is used in sufficient quantities, and with due regard to its dangerous qualities, it has proved of definite value in the cure of cancer. Since the total supply of the element is doubtless very small, it ought to be reserved for medical use. A great deal has, nevertheless, been employed in luminous paints, for watch dials and the like. These consist largely of a crystalline variety of zinc sulfide, with a trace of copper or manganese to serve as an activator and a trace of radium salt, though another radioactive element, mesothorium, has often been substituted for the radium.

About 1,000 Gm. of radium (in the form of radium salts) has been produced in all the world since the discovery of the element in 1898, and the present production is perhaps 100 Gm. a year.

The peculiar properties possessed by the radioactive elements—their ability, for example, to affect a photographic plate wrapped in black paper, to cause air to become a conductor of electricity, or to make certain minerals glow in the dark—were early found to be due to the fact that they emit invisible rays of radiations. These travel in straight lines, after the fashion of ordinary light, but are able to penetrate slight thicknesses of paper, wood, or even metal. As soon as a sample of a substance emitting these rays is placed in a magnetic field, it is observed that the rays are deflected, or bent aside; some toward the left, but others toward the right. This means that some of the rays carry positive electrical charges and others negative charges. A third type of ray, arising in many of the best-known cases of radioactivity, is quite unaffected by a magnetic field.

**Alpha-Rays.**—The three types of rays differ in other important respects. The first type (the alpha-rays) is now known to consist of doubly positively charged helium atoms ($He^{++}$), sometimes called *alpha-particles.* These are hurled off from the radioactive material with such enormous velocity (9,000 to 13,000 miles a second) that molecules of air with which they happen to collide are ionized (gaining or losing electrons, thus forming ions). In consequence, the air becomes a conductor of electricity. If the air has previously been saturated with water vapor, then suddenly cooled, each of the ions formed by the passage of an alpha-particle becomes the center of condensation for a microscopic droplet of liquid water. The path of each alpha-particle is thus made visible as a streak of fog.

**Beta-Rays.**—The second type of ray (the beta-rays) is shown to be negatively charged by the fact that it is deflected in the opposite direction from the alpha-rays when placed in a magnetic field. The beta-rays are now known to be streams of electrons, sometimes called *beta-particles,* moving in straight lines, with velocities from 16,000 to 180,000 miles a second. Electrons are very light, hence are very easily deflected by a magnetic field, and in very intense fields actually have their paths curled into spirals. These beta-rays are responsible for most of the photographic effect of radioactive substances. Beta-particles also produce fog tracks when they traverse air that has been supersaturated with moisture, but the tracks are wavering and uncertain, instead of being straight lines.

**Gamma-Rays.**—The third type of ray (the gamma-rays), being undeviated by a magnetic field, must be without electrical charges. These rays are a kind of radiant energy, even more penetrating than x-rays, and with a wavelength less than about a ten-thousandth of that of ordinary light. The most penetrating gamma-rays will penetrate many inches of solid steel, so they have occasionally replaced x-rays in the examination of steel castings in attempts to locate hidden flaws or slag particles.

**Pathology.**—According to Reznikoff,[15] radium, when ingested, acts like lead in its distribution, storage, and elimination. While much of the swallowed radium passes through the intestines unabsorbed, some enters the portal circulation and is carried to the liver. Most of the radium, 65 to 98 per cent, is eliminated within four days, 90 per cent in the feces and 10 per cent in the urine. Bile also contains radium, therefore affording a means of excretion. The unexcreted radium is removed by the phagocytic cells in the reticuloendothelial system. Eventually the radium is stored in the bones, at first in the trabeculae. It may be eliminated slowly in the stools and urine and a small proportion is lost by the expired air from the lungs as the gas radon. But this excretion accounts for a very small percentage of the total amount of radium in the body. To eliminate 1 microgram would require three to five years, and forty-five years would be necessary to rid the body of half of the quantity found in the average case of radium poisoning. Meanwhile the alpha-rays of the remaining radium continue their destructive bombardment. As little as 1 microgram or one-millionth of a gram distributed over the entire skeleton may cause death years after ingestion. The fate of ingested radium can be followed more accurately than can any other toxic substance because of the delicacy of electroscopic measurements and the help afforded by self-made photographs of the tissues.

**Signs and Symptoms.**—Radium is a slow, insidious poison. Anemia of the hyperchromic type may be the first evidence of radium poisoning, which later on very often assumes the aplastic form. Necrosis of various bones has been noted. Weakness, loss of weight, anorexia and other symptoms of anemia in general will be found in cases of radium poisoning.

**Treatment.**—There is no specific treatment if the radium has become stored in the body. Temporary relief from the anemia may be obtained by transfusions of whole blood. Victims of radium poisoning should avoid trauma to the bones.

It is industry's obligation to prevent radium poisoning, which need never occur if proper precautions are taken. But these measures must be carried out meticulously since radium enters the body with considerable ease. Prevention should be directed against the inhalation of the dust.

There are two groups of workers in the manufacture of radium dials who must be protected. The first of these are the painters—usually young women. These should be provided with individual glass hoods within which the operation is performed. The glass protects the worker's face, especially the breathing zone, yet enables a clear vision of the working field. The booth should be exhausted by suction ventilation. The head should be covered, the clothing simple, and no personal articles, such as cosmetics, cigarettes, candy or food, permitted within the radium room.

Rigid personal hygiene must be enforced under supervision and not left to the individual's inclination. An excellent regime has been suggested by Evans,[16] as follows:

> "*Washing.*—The hands must always be thoroughly cleaned before eating or smoking. To remove paint from the hands, Dr. George E. Morris has tested a number of solvents. He has found most effective and least toxic a mixture of one part xylene, one part trichlorethylene, and two parts ethyl alcohol. This mixture should be rubbed onto the dry skin and removed, before it evaporates, with soap and water. The skin is then thoroughly dried and examined in a darkened room under ultraviolet light, which will reveal any residual luminescent material. If the first cleaning is not complete, as is often the case, the entire solvent and washing process is repeated until the ultraviolet lamp reveals no residual radium paint. In the dark room many of the new ultraviolet lamp bulbs will be found satisfactory. For example, the GE 4 watt RP-12, 360 BL lamp can be obtained in a convenient fixture (Grimes Mfg. Co., Urbana, Ohio) including a filter for removing visible light. If a more powerful but less portable source is desired, the GE type BH4 (sunlamp with filter to remove visible light) may be installed. These new lamps are more suitable than the argon glow lamp because the latter gives more visible light and less ultra violet radiation.
>
> "One wash basin, with solvent, soap, hot and cold water, and paper towels should be provided for every five workers. A waste receptacle provided with a swinging lid operated by a foot pedal should be installed for used towels. In very large plants one wash basin per ten to fifteen active workers may suffice, especially if rest periods and lunch hours are staggered. The washing and dark room areas are most useful if adjacent to one another. Local ventilation should be provided in both areas to remove solvent fumes.
>
> "A separate sink should be provided for the disposal of contaminated solvents, which have been used to wash radium compound from imperfect or discarded work."

The other group involved in the radium room is that composed of inspectors, foreman, and those who clean the room. These are likewise exposed to the dust and need just as careful supervision in hygiene.

In order to make certain that the worker is not absorbing undue radium, two routine procedures are in order. One of these is to do a complete blood count once a month. The other is to have the worker undergo breath radon measurements. The reader is referred again to the article by Evans[16] for details of this measurement.

In the further prevention of radium absorption it is felt that an adequate intake of calcium is of value. This is based upon the belief that radium metabolism closely parallels that of calcium. On the other hand, if a worker reveals the presence within the body of excessive radium, then excretion of radium is aided by a low calcium intake.

In chronic radium poisoning, a regime directed toward aiding the excretion of radium should be followed. It is suggested that a low calcium diet, parathyroid extract, and large doses of ammonium chloride be instituted. That such treatment will have much effect is to be questioned. Treatment of any existing anemia is also advisable.

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# CHAPTER XXVII

# METAL FUME FEVER

Metal fume fever is purely an industrial disease. It is characterized by a febrile reaction following the inhalation of finely divided particles of the metallic oxides. Among the workingmen it is known as brass chills, brass foundry worker's ague, metal ague, zinc chills, spelter shakes, etc. It is an old disease referred to frequently in the literature and needs but brief description. It is most important that its *brief duration* and *absence of aftereffects* be emphasized.

**Pathology.**—Actually there is no pathology resulting from this exposure, the reaction being physiologic. It baffled the early experimenters as to just why the fumes produced an illness when the dry powder of these metals did not. Injecting soluble zinc oxide, Lehmann[1] was unable to produce noticeable symptoms. It was suggested that the inhalation of fumes killed bacteria in the respiratory tract which, when absorbed, caused symptoms. Lehmann thought it might be that the inhalation killed off the epithelial cells, the proteids of which, when absorbed, produced the symptoms. Drinker[2] held that it was a physical difference; that the particles of ordinary oxides are large, settle quickly, and rapidly collect in clumps to adhere to the sides of the trachea. The fumes, on the other hand, are so fine that they pass rapidly through the upper respiratory tract into the lungs.

**Immunity.**—Immunity to these fumes may be developed, but when it occurs there is leucocytosis, functioning as a defense mechanism. It is noticed that if a workman absents himself from his exposure for a few days, he will lose this immunity. Many workers report a state of well-being during the week, but after a weekend they will notice various grades of reaction upon a return to work.

Certain workers have reported disturbances of liver function, glycosuria, and increased blood pressure and pulse rate.

**Signs and Symptoms.**—These are closely comparable to influenza or la grippe. Chills come on usually after the patient has left work and, last for one or two hours, rarely longer. The body aches all over; nausea and not infrequently vomiting may occur; fever sets in and the temperature may reach 102° F., seldom higher; the throat is dry, and there may be a dry cough. Weakness, lassitude, or exhaustion are complained of. The leucocyte count may range between 12,000 and 16,000 and may persist after all other symptoms disappear. A mild leucocytosis is present, as previously noted, in those without symptoms.

*Chronic symptoms* are rarely present other than in some instances when a complaint of general lassitude is made. Metal fume fever is acute in onset and short in duration, the patient usually making a complete recovery within twenty-four or thirty-six hours. Pronounced chronic symptoms should arouse suspicion of the likelihood that lead might be the etiologic factor.

**Medicolegal Aspects.**—

TEMPORARY DISABILITY.—This rarely lasts more than one or two weeks. In mild instances the patient loses no time from work, merely changing his job or receiving better protection. Patients in more acute cases usually return to work in several days.

PERMANENT DISABILITY.—Permanent disability as a result of metal fume fever is unknown.

**Treatment.**—

LA GRIPPE REGIMEN.—No specific therapy is indicated in these cases. Patients may be treated as though they had la grippe. The administration of acetylsalicylic acid, 10 grains (0.65 Gm.), every four to six hours, combined at times with codeine phosphate, from ½ to 1 grain (0.0325 to 0.065 Gm.), will afford relief. The forcing of fluids (water and fruit juices), the use of hot drinks, and rest in bed are of value. These patients will become symptom free without treatment after absence from exposure for a short time, but for their composure the regimen given is prescribed.

PROPHYLAXIS.—The most valuable point, however, is to adopt an adequate medical and engineering program for prevention of the metal fume fever.

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# PART FOUR

# THE DUSTS

The dusts in industry are ubiquitous and of many types. Certain dusts such as lead or manganese may cause systemic poisoning, while others may act as allergens, irritants, or corrosives. At this time, interest is focused on the nontoxic dusts which are either organic or inorganic.

It is a common practice in most textbooks on industrial medicine to allot considerable space to the action and pathology of two inorganic dusts, namely, silica and asbestos, but to dismiss the organic dusts with the brief remark that they cause no pulmonary disease. Yet, such a curt dismissal is hardly sufficient in view of the fact that the profession at large has not been taught to differentiate the organic from the inorganic dusts, nor is the average physician adequately informed as to why the organic dusts are comparatively inert.

Consider for a moment an environment where the workmen are using wool glass or fiber glass. Small particles of these materials get into the skin, causing irritation. Is it not reasonable for the workmen to believe that they also inhale this substance and that it can cause harm to the lung—an assumption frequently shared by the physician. The same apprehension is likely to arise regarding cement dust, steel dust, saw dust, particles from a grinding machine, and many other sources. Therefore, an explanation of the reason why these dusts cause no harm is obligatory in a textbook of this type. This explanation will be given in the chapter devoted to the inert dusts.

It is also necessary to clarify the significance of pulmonary fibrosis. It is conceivable that a prolonged exposure (in terms of years) to many organic dusts could lead to some degree of fibrosis of the lungs. But a fibrosis in itself is not essentially disabling. Even marked fibrosis in advanced silicosis, uncomplicated by infection, is rarely disabling to a high degree. It is important that physicians should not confuse fibrosis of the lungs with disability.

Finally, the word pneumoconiosis needs to be redefined. Time and usage have induced the erroneous belief that this term is synonymous with silicosis. Modern authorities no longer hold to the interpretation that pneumoconiosis means *necessarily* a chronic fibrosis of the lungs. Sander[5] correctly defines pneumoconiosis as "dust added to the lungs." This definition implies that certain dusts such as silica or asbestos may cause fibrosis but that others cause only exaggerated peribronchial markings or a harmless pigmentation, such as induced by iron dust (siderosis).

In this presentation of the dusts, pulmonary pathology due to the infestation of certain fungi has been omitted. It is recognized that certain occupations expose workmen to a variety of molds or fungi and these have been mentioned in discussing certain of the pneumoconioses. However, any detailed consideration must be left to the mycologist.

# CHAPTER XXVIII

## SILICOSIS

Before presenting the clinical and pathologic picture of silocosis, a review of the chemical nature and the geologic origin of silica is in order. Such a review will serve to distinguish silicon dioxide, or free silica, from the silicates and will also reveal the variations encountered in the silicate industry.

According to Deming[1] the element silicon is second only to oxygen in abundance in the crust of the earth, of which it constitutes about 28 per cent. It never occurs free, but only in combination with oxygen or with oxygen and metals, in the silicate minerals, which compose most of the earth's crust.

Quartz is a crystalline form of silicon dioxide, $SiO_2$, commonly termed *silica.* It is formed by slow deposition from solution in water, during the lapse of centuries. At other times the silica separates from solution as a colorless gel, which is gradually dried and compacted into flint.

The solid rock of the earth's crust has an average composition approximating salts of metasilicic acid, $H_2SiO_3$. But many individual rocks depart widely from this average. Rocks, for the most part, are not definite chemical compounds, though if they have cooled slowly enough they may contain crystals which are compounds embedded in a glassy ground mass (supercooled liquid). Thus *granite* consists of three different types of crystals.

The chemical compounds that go to make up the *igneous rocks* (namely, those derived from originally molten material by a process of crystallization) are called *primary minerals.* In addition, there are a great many derived or *secondary minerals,* produced by alternation of the primary minerals, through heat, pressure, or circulating water. Thus feldspar, the principal component of granite, may be altered to mica and quartz by heat and pressure, or may produce clay by weathering.

Living organisms of the humbler type sometimes employ calcium carbonate and sometimes silica as a skeleton or supporting structure. The higher forms of life in the ocean are chiefly supported by marine vegetation that is largely made up of very minute plants called *diatoms,* having a covering of silica. The diatoms that throng in every fresh-water pond are of fascinatingly beautiful and varied shapes. A liter of sea water often contains several million of these.

**Silica in Industries.**—Silica brick, made of nearly pure silica, sintered into solid blocks with the addition of very little clay to serve as a binder, is extensively used as a refractory for furnace linings, at temperatures at which ordinary brick would soften or melt. Silica brick has the advantage of a lower coefficient of expansion than ordinary brick; that is, it expands less when heated to high temperatures. Silica brick at high temperatures must be protected from the action of metallic oxides and "basic" slags (those rich in metallic oxides), since these materials react with silica to form fusible silicates. But silica brick resists highly silicious slags and molten glass, hence may be used for lining furnaces for manufacture of glass, glazes, and enamels.

Finely divided silica slowly reacts with alkaline solution, even at room temperature, forming soluble silicates, or the particles of silica may become coated with a gelatinous hydrous silicate, so that the mixture presently sets to a hard mass. When finely divided silica is mixed with lime (CaO) and exposed to steam, the mixture sets to a hard mass, a hydrated calcium silicate. This reaction is the basis of an important industry—the production of sand-lime brick.

Silica melts at 1,670° C., a temperature far above that reached in any gas or coal-fired furnace, but readily attained in an electric furnace. Chemical apparatus of fused silica, which is manufactured by melting sand in an electric furnace, is of importance in the laboratory as a substitute for platinum. It is remarkable for its resistance to acids but is fairly readily attacked by alkalies. Its rate of expansion with changing temperature is only about one-fortieth that of glass. Most fused silica is milky because of bubbles of air, but when it is made from clear quartz crystals, or when white sand is melted in a vacuum furnace to remove the air, the produce is quite transparent.

**Asbestos and Talc.**—Asbestos is any one of several fibrous silicate minerals. The two most important varieties are hydrated magnesium silicates. One of these is short fibered but very infusible and acid resistant. It is accordingly used for packing steampipes and furnaces and is used in the chemical industries for filtering acids. The longer-fibered, less heat-resistant variety, coming principally from eastern Canada, is woven into fireproof fabrics. The fibrous structure of asbestos results from a linking of the silicate tetrahedra to form long chains. A bundle of these chains, with cations intervening between them, constitutes an asbesos fiber.

Closely related to asbestos is *talc*, also a hydrated magnesium silicate. This is a white mineral with a flaky texture. It is soft enough to be scratched with the fingernail, and so smooth as to be almost greasy to the touch. It is used in cosmetics, for filling writing paper, for finishing leather, and for polishing glass and metals. *Soapstone*, used in electrical switchboards, and for sinks and table tops, is a relative of talc. The United States produces over 100,000 tons of the two minerals each year.

**Mica.**—Mica is a familiar transparent mineral which cleaves into thin flakes. This is muscovite, or potash mica, a hydrated potassium-aluminum silicate. Another variety of mica is used between the commutator bars of direct current electric machinery. It is suited to this purpose because of its high electrical resistance and because it has practically the same hardness as copper, and hence wears down at the same rate as the commutator bars between which it is placed.

**Sodium Silicate.**—When a mixture of sand and soda (sodium carbonate) is strongly heated, the mixture evolves carbon dioxide, becomes pasty, and presently fuses to a clear liquid. If we pour this out on a stone slab, it soon hardens to a glassy mass of sodium silicate.

The proportion of sand to sodium carbonate may vary widely. It seems as if this violates the law of constant composition. The explanation is that the product we call sodium silicate is not a single compound but a mixture of several compounds, dispersed in one another—in brief, a solid colloidal dispersion.

Sodium silicate, thus prepared, will not dissolve very readily in cold water; but if the white hot, molten material, obtained by fusing sand with sodium carbonate, is poured directly into water, it is dispersed to form a viscous colloidal dispersion called "water glass." This is an important adhesive for sealing cardboard cartons, for when the water evaporates it leaves behind a glassy mass which it is practically impossible to redisperse.

Eggs will keep for months if submerged in diluted water glass, which is sufficiently alkaline to discourage bacteria. Moreover, it tends to exclude air from the pores of the eggshell. The faintly alkaline nature of sodium silicate enables it to be used as a filler in laundry soap.

**Silica Gel.**—If a little acid is added to a dilute solution of water glass, the liquid usually stiffens at once or within a few minutes to a transparent gel, which contains as much as 98 per cent water. This product is sometimes called colloidal silicic acid. A better name is *hydrous silica.* By exposing this gel to a dry atmosphere, or even by squeezing it in a press, it gradually loses water and is converted into a porous, dry mass of silica. This material, when prepared in such a way as to retain its porous structure after drying, is known as *silica gel.* Its remarkable capacity for absorbing gases and vapors is put to use in certain types of refrigerators, in removing gasoline vapors from natural gas, and in other industries.

**Lime-Soda Glass.**—Any material which breaks with a characteristic wavy fracture and possesses a glassy luster, with some degree of transparency, is often referred to loosely as a glass. Ordinary soft glass, or lime-soda glass, used for window panes and bottles, is a sodium-calcium silicate; but useful glasses have been prepared containing none of the three elements Na, Ca, Si.

The most important raw materials for the manufacture of common glass are calcium carbonate, sodium carbonate, and quartz. These must be very finely powdered and intimately mixed. If the glass is to be colorless the raw materials should be free from more than traces of iron. When such a mixture is heated in a furnace, the silica reacts with the carbonates, expelling carbon dioxide. After a few hours the contents of the crucible melt down into a clear liquid, which is purified by skimming, then cast or blown into molds.

In addition to the industrial uses of silica just mentioned, exposure also exists in the following occupations: mining of hard silicious rock (copper, gold, silver, zinc, iron, and hard coal); quarrying of granite, sandstone, quartz, slate rock, and crystal; pottery making, ceramic industries; glass manufacturing; working with talc and abrasive material (sand, sandstone, sandpaper, scouring material, and soaps); brick manufacturing when silica is used; stone finishing; construction work (tubes, tunnels, aqueducts, etc.); sand blasting.

**Definition of Silicosis.**—"Silicosis means a disease of the lungs due to breathing air containing uncombined silicon dioxide dust, characterized anatomically by generalized nodular fibrotic changes throughout both lungs, which are demonstrable by x-ray examination and by autopsy, and resulting from any process of occupation involving inhalation of silicon dioxide dust." (Gardner.[3])

According to Lanza and Goldberg,[2] the factors necessary to produce a case of silicosis are (1) the amount of dust in the air at the working place and breathed by the workman; (2) the percentage of free silica in the dust; (3) the duration and constancy of the exposure; and (4) individual susceptibility. These factors may be enlarged upon as follows:

**Size of the Dust Particle.**—It is believed by many that only free silica having a size range up to 3 microns is capable of producing pulmonary fibrosis. It is frequently stated in the literature that particles may be dangerous in a range up to 10 microns, but there is evidence that the majority of particles entering the lungs are much smaller, probably 5 microns or less.

**Permissible Concentrations.**—The safe limit of concentration in which a man can work in free silica is not definitely established. Since certain areas of work contain more free silica in the material than others, the permissible limit will vary. For instance, in the South African territory, the percentage of free silica in the dust is 80 per cent, and therefore the permissible concentration of million particles per cubic foot has been set at 4.5 whereas in the Pennsylvania anthracite district the free silica is estimated at 35 per cent and the permissible limit is 5 to 10 million particles per cubic foot.

It is also believed that certain minerals present in the atmosphere together with free silica modify the action of the silica. Some of these are aluminum, carbon, hematite, etc.

**Length of Exposure.**—As will be mentioned elsewhere, it is generally held that at least ten years are required for a significant degree of silicosis to develop. Exceptions to this observation have been pointed out under the History of Exposure.

**Diagnosis.—**

History of Exposure.—Clear-cut and indisputable evidence of silicosis by x-ray leaves no doubt as to the occupational cause of the disease. But in those cases where the fibrosis is mild to moderate, the lesion may be, and often is, due to some cause other than silica. In such instances the patient's statement that he has been working in a silica-laden atmosphere cannot be accepted without confirmation.

Disabling exposure consists of working in an atmosphere of free silica over a period of years. A significant degree of silicosis rarely develops until after ten or more years of exposure. An exception to this rule has been noted in sandblasters or drillers in unventilated tunnels where the rock has been high in silica content, but such instances have been rare and startling. Silicosis is a cumulative condition and time is necessary for irritation to result from tissue reaction. Silicosis does not result from short exposure or "from a dust storm, or driving on dusty roads . . . It is not produced by a few weeks employment in the dustiest mine or mill that can be found."[3]

**Signs and Symptoms.**—The initial effect of a collection of silicotic nodules within the lung is attended by no symptoms. Many persons may develop what appears to be intense nodulation and fibrosis without evidencing any signs or symptoms during their life. Râles will not be heard, the chest will usually be barrel-shaped, and inspection will generally show some bilateral limitation of excursion. Otherwise these persons appear normal. Even respiratory diseases of another nature, such as pneumonia or bronchitis, may be overcome without aggravating silicosis.

Progressive Symptoms.—If the patient does show symptoms, the first is that of shortness of breath upon minor effort. This increases in severity, and palpitation of the heart may be noted. At about this time, a dry, infrequent cough may ensue, which, as time goes on, becomes moist and frequent. The sputum is usually scant, stringy, and sometimes discolored. Fever is conspicuously absent. Unaware of his true condition, the patient may request lighter work. Further progression may include pleurisy pains, epigastric pains, loss of appetite, and marked fatigue. As dyspnea increases, cyanosis appears. Progressive heart failure may continue until death, but these cases are rare. At this late stage, tuberculosis may enter the picture. This condition, silicotuberculosis, will be described later.

X-RAY DIAGNOSIS.—The most important evidence of the presence of silicosis and its degree is to be found in the x-ray films. In a case of questionable etiology, a differential diagnosis should utilize both stereoscopic and lateral views. In the differential diagnosis the interpretation of the films must rule out passive congestion of the lungs from cardiac decompensation, malignant disease, and mycotic infections.

Pancoast and Pendergrass[4] tabulated the roentgenologic and histologic appearance of silicosis, and silicosis with infection, as shown in Table XVII.

TABLE XVII. SIMPLE SILICOSIS

| ROENTGENOLOGIC APPEARANCES | HISTOLOGIC APPEARANCES |
|---|---|
| Nodulation; discrete shadows not exceeding 6 mm. in diameter tending to uniformity in size, density, and bilateral distribution, with well-defined borders surrounded by apparently normal lung shadow; outer and lower lung fields characteristically show fewer nodules | Circumscribed nodules of hyaline fibrosis located in parenchyma of lung; occasionally some of these nodules may show microscopic foci of central necrosis |
| Conglomerate shadows that appear to result from a combination or consolidation of nodulation usually with associated emphysema manifested by:<br>(a) Localized increased transparency of lung with loss of fine detail<br>(b) Intensification of trunk shadows by contrast<br>(c) Depression of domes with possible tendency toward individualization of the costal components of diaphragm<br>(d) Lateral view; increase in pre-arotic and retrocardiac spaces with exaggerated forward bowing of sternum and backward curving of the spine; widening of spaces between ribs may or may not be present | Result of coalescence of discrete nodules; an area in which the nodules are closely packed and most of the intervening lung is replaced by more or less hyaline fibrous tissue; lung architecture particularly obscured; no demonstrable evidence of infection; emphysema is a compensatory dilatation of air spaces with or without thickening of septa |

Table XVII should be augmented by a more detailed description of the chest roentgenograms as described in one of the last papers read by the late beloved Leroy Gardner.[3]

"Chest roentgenograms reveal patterns that vary with the character of the pathologic changes in the lung and these in turn vary with the kind of dust that has been inhaled. Dust reaction in a normal lung will have different characteristics from that in lungs with associated infections in either healed or active state. While these patterns are by no means definitive for particular dusts, they are sufficiently diverse to permit classification into certain main types. It is the writer's conviction that the response to dusts of different types is so widely diverse that it serves no useful purpose to include every manifestation of all the pneumoconioses in a common classification. To do so may unintentionally imply progression from one phase to another that is quite beyond the potentialities of the condition under consideration. Silicosis, the reaction to free crystalline silica, is recognized pathologically by multiple, small nodules of fibrous tissue distributed uniformly throughout both lungs. Asbestosis, the reaction to silicates in *fibrous form*, is similarly identified by a fine, tenuous, diffuse fibrosis which replaces large amounts of functional lung tissue. Most other dusts that have been well studied, including a majority of the nonfibrous silicates, cause major reactions in the walls of blood vessels and perhaps the septa between lobules of lung tissue. A few may have secondary effects on the walls of the functional air spaces but these forms are still under investigation. These different kinds of pathologic changes cast shadows on an x-ray film that group themselves into four main pattern groups. To facilitate description the common categories will be cataloged and described with comments upon their significance.

"*Categories of X-Ray Shadows.*—1. Simple exaggeration of the normal linear markings. Most of the shadows cast by x-rays traversing a normal lung are due to vessels containing blood. The air-filled bronchial tree throws no shadow except in the case of the stem bronchi and trachea whose thick walls are barely visualized in a good roentgenogram. The shadow pattern of the normal lung is therefore comparable to that of a leafless tree with the finer twigs at the periphery not clearly seen. Any change which thickens the pulmonary vessels will result in heavier vascular shadows involving both large and finer branches. It so happens that most inhaled materials tend to accumulate first in the walls of the pulmonary vessels. The mere aggregation of particles themselves or the slight amounts of reaction that they provoke increases the density of the delicate connective tissues about the vessels. When such changes are at all marked the shadow of the vessel is heavier than normal. As a great many kinds of dust produce such changes, exaggerated linear markings are common findings in persons employed in industry. There is nothing specific about such a pattern to indicate its cause and it is rarely associated with clinical symptoms. It may be the precursor of subsequent silicotic changes but this cannot be inferred because in many persons, regardless of exposure, no further development follows.

"2. Reticulation is a term recently popularized in England as descriptive of the shadow pattern in the films of coal miners. In this country certain mixtures of silicate dusts, including some of the talcs for example, have produced similar effects. Between the exaggerated linear shadows of the thickened blood vessels is a background of small, ill-defined ringlets which in the aggregate suggests the appearance of a rough orange peel. Apparently these shadows are cast by thickened septa between lobules of lung tissue. Like the outer coats of blood vessels the coarser septa of the lung contain large lymphatic trunks and some kinds of dust particles tend to accumulate about them. The exact reason of such localization with only certain kinds of dust is not clear. Symptoms are said to be more frequent in persons with reticulation than with simple linear exaggeration, but further study of these subjects is necessary to be certain whether this is invariably true. Disability from this cause is hard to understand unless the thickenings of the septa reduces the elasticity of the lungs.

"3. Nodulation is the term used widely in this country to describe the small, round, discrete shadows cast by the nodules of fibrous tissue that constitute the characteristic tissue reaction of silicosis. In typical cases of moderate severity occurring in lungs not altered by associated infection, the nodular shadows, not over three or four millimeters in diameter, are uniformly distributed throughout all parts of both lungs. In long standing silicosis the costo-phrenic portions of the lung fields are usually clear due to the development of compensatory emphysema incident to retraction of the upper lungs. There may also be a limited tendency to conglomeration of nodules in *small* areas but on stereoscopic examination the outlines of the individual units are still discernible. Such small conglomerations are more apparent than real, and are often due to fortuitous superimposition of shadows viewed on a flat film. The larger, massive conglomerations in which the outlines of component nodules are no longer seen are probably due to associated infection and will be discussed later.

"When silicotic nodulation begins to make its appearance in roentgenograms the branching shadows of the blood vessels are blurred, and later as the nodules become larger, they are completely obscured. These developments have been compared first to the shadow cast by a tree in bud and then by one in full leaf.

"While there are other conditions which may simulate the roentgenographic picture of silicosis, the shadow pattern described when taken in conjunction with the clinical story and a history of adequate exposure to free silica dust constitutes strong evidence for a positive diagnosis. A few other dusts like the exceedingly fine metallic iron in welding fumes and other rare substances may produce similar effects, but then the occupational history becomes decisive. Reactions to infection lack the uniformity of distribution of silicosis and the patients are obviously ill; tumor metastases rarely exhibit the uniformity of size and distribution of the nodular reaction to free silica.

"To indicate the severity of the silicosis, it is customary to classify the x-ray films by numerical stages—1, 2 or 3 depending upon the number and size of the nodules. While certain individuals examined in serial films show evidence of progression through these successive stages this is not invariably the case. Furthermore, a stage 1 lesion in a granite cutter is apt to be quite different in appearance from a stage 1 in a hematite or a gold miner from some other location. The components of the dust other than quartz account in large part for local variations in the picture. A subdivision of silicotic shadow patterns into stages is useful in indicating progression of disease within a particular group but it is doubtful whether it has much value in comparing the severity of the silicosis in different locations.

"4. Massive conglomerate nodulation. The writer strongly subscribes to the South African view that the presence of large localized areas of conglomeration in a film showing evidence of nodulation constitutes evidence of associated infection in the lungs. The infection, in an active, dormant or healed state, has injured a localized portion of lung tissue so that more silica is retained in the area. Unusual numbers of nodules form locally and furthermore there is stimulation of any connective tissue formed in response to the infection itself. The result is a mass of nodules embedded in a solid matrix of silicotic scar. The x-ray casts a solid shadow which the stereoscope does not resolve into its individual components. Such massive conglomerate lesions generally occur in the upper portions of the lung. They may be bilaterally symmetrical; they may radiate outward from the hila. Sometimes they involve the lower lungs.

"In early or moderately advanced conglomerate disease the rest of the lung fields exhibit the picture of nodulation. In old cases, however, contraction of the massive scars, which are usually covered by fibrous pleural adhesions, results in marked distortion of the lungs. Most of the more normal tissue with its contained nodules is pulled into the orbit of the massive scar and then bullous emphysema overdistends the remaining part of the lung. In such cases the usual pattern of generalized nodulation is disturbed and perhaps only a few or even no discrete nodules can be visualized. Here diagnosis must lean heavily on the history of occupational exposure.

"The nature and character of the underlying infection is hard to establish. Tuberculosis is much the most common cause and this etiology can be proved in about 60 per cent of the lesions studied postmortem. Other organisms capable of producing an organizing pneumonia could have the same effect. As will be noted later, the problem of determining activity of a tuberculous component of one of these massive conglomerate lesions is most difficult for a sterile scar can cause almost as much damage as scar tissue encapsulating a nidus of living tubercle bacilli. Years later these bacilli buried deep in silicotic fibrosis may finally reach its surface and when they do death from rapidly progressive tuberculosis is imminent. Repeated films taken at intervals of a year may show no apparent change, but as soon as the size or shape of the area is altered then suspect activity of the underlying activity.

"5. Diffuse haze to ground glass appearance. The former appearance was one described as a precursor to the development of silicotic nodulation, but in recent years it is less frequently mentioned. It is conceivable that such haziness might result from exposure to exceptionally severe industrial exposure, as a result of which the air spaces of the lungs would contain enough dust phagocytes to intercept x-rays and thus cast a shadow. Today this type of abnormal shadow is most commonly associated with early asbestosis and a few of the less known pneumoconioses. In early cases such shadows spread over the lower lung fields to more or less obscure the outlines of the normal vascular markings. As the disease advances the haze becomes more dense and extends upward toward the apices. In fully mature asbestosis all vascular markings throughout the lower two-thirds of the lungs are obliterated by the so-called ground glass appearances, a widespread, uniform density composed of confluent, minute stippled shadows.

"In the diagnosis of asbestosis such patterns must be associated with a history of exposure to *fibrous* asbestos dust usually for periods of six to eight years or more. Asbestos is often mixed with other minerals for industrial use and unless there is a sufficient proportion of long fiber in the dust generated from the mixture the x-ray pattern just mentioned will not be produced. Inhalation of particulate matter may result in linear exaggeration but this cannot be diagnosed as asbestosis.

"*General Considerations.*—The film of a person with a history of exposure to dust in industry should be viewed with the same objectivity as those of any other patient, and one should studiously avoid the conclusion that every abnormality detected is due to the occupational exposure. Localized shadows involving one lung are practically never due to inhaled dust; infection is much more probable. Miliary calcification, a condition of unknown etiology, is not uncommon in industrial workers. The lung fields are studded with small, hard, round shadows but unlike the nodulation of silicosis, these lesions vary in size and distribution and only by occasional coincidence is there a history of exposure to siliceous dust. Tumors and miliary tuberculosis can be eliminated by the condition of the patient and lack of uniformity in distribution of nodules. Linear exaggeration is a nonspecific effect of anything which thickens the blood vessels or their connective tissue sheaths. When the change is well marked and there is an associated history of dust exposure the diagnosis of pneumoconiosis is tenable. Heart failure leaves its marks in the lower lung field; advancing age causes some accentuation of the vascular pattern. Finally it should be remembered that a poor film or one taken short of full inspiration produces artefacts that are often mistaken as evidence of pneumoconiosis. Full size 14 x 17 inch films are none too good for the purposes in hand and stereoscopic films are still better as they throw shadows into perspective and thereby tend to minimize the severity of abnormal conditions. Where possible comparative study of films from other employees in the same industry is immensely helpful in establishing the characteristics of a particular pneumoconiosis. It cannot be done from an isolated case. And finally it must be appreciated that serial films taken at yearly intervals on a group of individuals will teach an observer more than all that has ever been written on the subject."

**Pathology.**—The pathology of silicosis is one involving the lymphatic drainage of the lung. The normal lymphatic drainage is toward the hilum. A small area beneath the visceral pleura is drained into the subpleural cistern and from there along the lobar septa to the hilum. Dust which reaches the wall of an alveolus is picked up by the phagocytic cells, and by ameboid movement they pass into the lymph spaces. From there they slowly move on to the minute lymph islands which guard the entrance to the small lymphatic vessels, pass through these and are finally picked up by the lymph glands at the hilus of the lung. It is believed that at this point the dust cells, acted upon by the alkaline body fluids, form a colloidal silica hydroxide which is a strong protoplasmic poison, and its action causes death to the dust cell and necrosis to the immediate surrounding tissue. Scar (fibrous) tissue is formed, which acts as a blockage to the lymphatic channels, and the removal of further inhaled dust becomes impossible (Figs. 37 to 40).

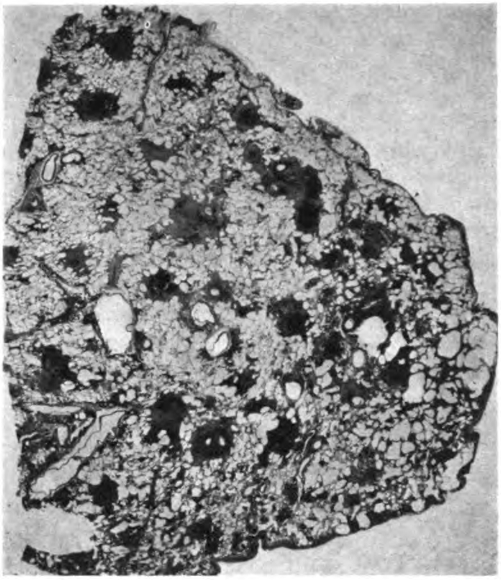

Fig. 37.—Simple discrete nodulation of uncomplicated silicosis. (Gardner.)

**Spread of Fibrosis.**—Additional fibrous tissue forms along the lymphatic vessels which accompany the blood vessels of the lung, invades the septa between the lobes, and spreads into the lung tissue itself. The minute masses of lymphatic tissue become fibrotic nodules and these, as time goes on, increase in number to block off large portions of the lung.

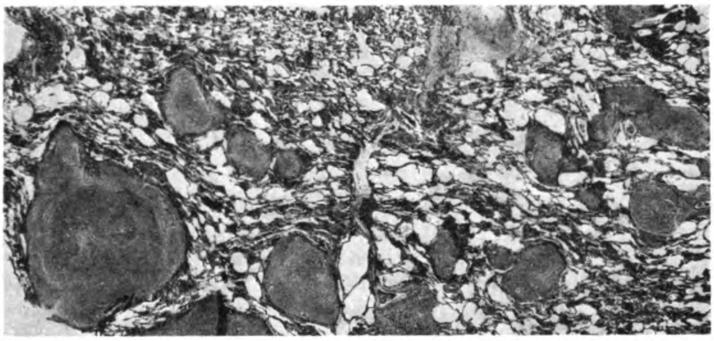

Fig. 38.—Massive areas of confluent nodules. Note the thickened pleura. (U. S. Public Health Service, Bulletin 241.)

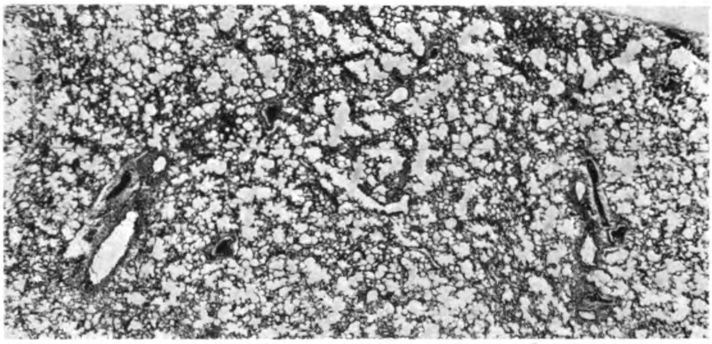

Fig. 39.—Confluent mass of silicotic nodules adjacent to small bronchus and blood vessels. Note the presence of extensive emphysema in patent lung tissue. (U. S. Public Health Service, Bulletin 241.)

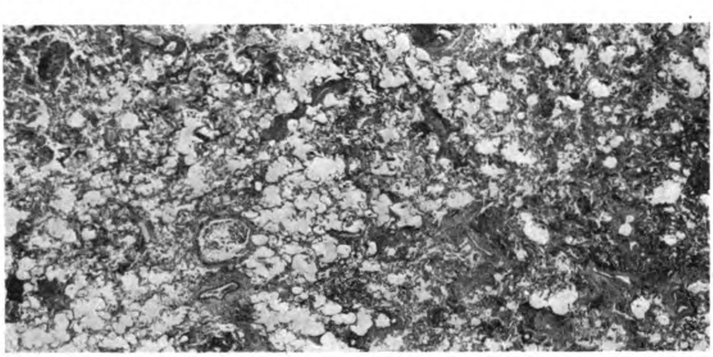

Fig. 40.—Section through lung, showing massive areas of confluent, fibrous nodules. Close inspection will reveal the extensive emphysema. (U. S. Public Health Service, Bulletin 241.)

This blocking of the lymph flow toward the hilus increases the spread of dust cells toward the pleura. These minute nodules of fibrotic material are thus scattered throughout the field and are surrounded by apparently normal lung tissue.

**Alveolar Consolidation.**—As the process proceeds they tend to coalesce to form massive areas of fibrosis. This increase of fibrosis tends to destroy the alveoli and thus provokes enlargement of the surrounding alveoli. Such enlargement constitutes emphysema. With the thickening of the alveoli, the elasticity of the lung is interfered with, the aeration decreases. If compensatory emphysema is adequate and infection is not present, the condition is that of simple silicosis. But if compensation is not adequate, the first symptom of silicosis appears; namely, dyspnea. However, since only one-fourth of one lung is necessary for life, fibrosis must be enormous before death can occur from deficient aeration alone.

**Production of Dyspnea.**—That encroachment upon the normal lung fields by nodulation is the cause of dyspnea is questioned by Cole and Cole[6]:

> "It is universally conceded that small, hard, dense nodules in the lung are pathognomonic of silicosis, yet these small, hard, dense, nodules are either absent or relatively infrequent in some cases of advanced silicosis in which dyspnea is an outstanding symptom. These silicotic nodules as observed roentgenologically often progress to an extreme stage before they cause dyspnea, yet dyspnea may occur and terminate fatally without a preponderance of the typical well-defined nodules. Spherical whorls or nodules are considered pathognomonic of silicosis, yet we believe they constitute a protective mechanism—a constructive rather than a destructive process—and are not responsible for the outstanding symptom of silicosis, dyspnea.
>
> "Advanced silicosis is frequently found only by accident, when the patient is being examined roentgenologically for some other lesion or injury, as a fractured rib. Such accidental discoveries are frequent, but, on the other hand, roentgenograms may show no evidence of typical nodulation even in a case of advanced silicosis."

**Silico-Tuberculosis.**—It is generally accepted that the commonest cause of disability and death in the patient with silicosis is tuberculosis, although it is not clear why the patient with silicosis becomes tubercular. As to whether the frequency of silico-tuberculosis is greater in those harboring tubercle bacilli prior to their exposure or in those who become infected after silicosis has transpired has not been determined. It is, however, known that the presence of the silica in the lungs tends to make the infection active and chronic. In a study of tuberculosis and its relation to silicosis, Sander[5] examined a large group of workers in Wisconsin, some 15,000 of which comprised the major portion of the dusty trade employees of the Milwaukee area. From his observation he states:

> "In the majority of cases of silico-tuberculosis which we see, the lungs undoubtedly became infected with an adult lesion before age 40 and the first effect of the silica inhalation was to aid in localization of the infection. Whether infection occurred before dusting began or after makes little difference at this time on the course of the case. The accumulating silica tends to make the infection extremely chronic, throwing around it a dense wall of fibrosis, such that clinical evidence of activity is entirely lacking for many years. Some of these cases may have had their lives prolonged by reason of their silica exposure, but most of them probably would have healed their infection spontaneously had they not been exposed to dust. It is this effect of silica which is the deleterious one, namely, the tendency to keep the bacilli alive and aid in their multiplication as more and

more silica accumulates in the lungs. In other words, the lesions are kept pathologically active by the silica, so that in the later decades when silicosis has become established and the protecting wall of fibrosis breaks down, a massive spread of the infection occurs, followed by clinical signs of its activity.

"It should be emphasized that this concept of the silica effect on the tuberculosis mortality is stated as being the usual one in our experience, but it is not necessarily the rule in all cases. It is not possible to be too dogmatic with such a disease as tuberculosis, particularly when it occurs in the form of silico-tuberculosis. That the latter is a true disease entity of itself and not merely a summation of the effects of silicosis and tuberculosis has become more firmly impressed in our minds year after year. It is so different from either tuberculosis or silicosis alone that neither is recognizable as a separate entity in some cases."

When tuberculosis complicates silicosis in an individual whose exposure has not been severe and in whom the degree of silicosis is mild, the course of the combined diseases may be chronic, the individual living for a number of years with death eventually resulting from tuberculosis. But where the exposure has been severe the course is usually rapid. The victim develops fever, loss of weight, profuse expectoration, and weakness. Death invariably occurs in one to two years after the infection becomes clinically evident.

The x-ray characteristics of silico-tuberculosis have been described. The pathologic changes commence with rapid increase in the size of the fibrous nodules. This is followed by the development of an interstitial fibrosis in the area of the lung tissue between the enlarging nodules. With the coalescence of these individual nodules, the conglomerate result is indicative of inflammatory reaction. These coalescent masses together with dense fibrous tissue are usually found in the upper and middle zones. Cavity formation, typical of tuberculosis, is not commonly found in silico-tuberculosis.

When silicosis is complicated by tuberculosis, the visceral pleura is thickened and marked scarring of the pleura is noted. In the advanced stages, masses of adhesions are often seen between the visceral and parietal layers, Pleural effusion is not common.

**Other Complications.**—Emphysema is a constant accompanying condition of advancing silicosis. Regarding pneumonia, the evidence in recent years is to the effect that silicosis does not predispose to pneumonia. There is equally no evidence to the effect that silicosis predisposes to carcinoma.

Many persons with silicosis apparently die of cardiac failure although post-mortem examinations rarely reveal any involvement of the myocardium or heart valves. It would seem that years of cardiac effort against resistance could produce myocardial weakness. Electrocardiograms of persons with silicosis show no significant changes.

It has generally been accepted that dyspnea is due to an increase in the amount of fibrous tissue and to inelasticity of the lungs, preventing proper ventilation. Cole and Cole[6] believe that the dyspnea attendent upon silicosis is due to capillary occlusion in the large localized regions of the lungs, with compensating capillary dilatation in other regions. Dernehl and Nau[7] suggest that silica acts as a bronchoconstrictor and that dyspnea is due to bronchospasm.

**Additional Aids in Diagnosis.**—Although the history of exposure and the x-ray pattern are the two chief sources for determining the diagnosis, there are occasions when an estimation of the silicia content of the dust will be necessary. In such instances recourse may be had to a chemical method, to petrographic identification, or to x-ray diffraction. The description of these three methods is too detailed to be included in a book of this kind, although some consideration will be given to these methods in the discussion of Case 2. The references at the end of this chapter list several articles which consider these methods in detail.[6, 8]

**Disability.**—When silicosis is complicated by tuberculosis, the victim should receive the full benefits of workman's compensation for a permanent total disability.

The estimation of partial disability is difficult, and no method has been evolved which is practical for general use. Nor does there exist general agreement to the effect that an individual with partial disability should be awarded compensation. The legal and medical aspects of "partial disability from silicosis" were discussed at the Ninth Annual Meeting of the Industrial Hygiene Foundation. Representing the legal phase of the problem, Waters[10] summarized the arguments on either side of the question as follows:

"*First. Reasons for Denying Compensation:*

> "1. Impossibility of evaluation of extent of disability. The paper prepared by Dr. Wright deals with the medical aspects of determining partial disability in silicotics and it is not necessary for me to give further emphasis to that part of the discussion. However, until there is some method that has the approval of the medical profession for the evaluation of partial disability in silicotics it would seem desirable to deny compensation for partial disability.
>
> "2. Most employees exposed to dust hazards show some fibrosis of the lungs and every employee working in a dusty trade would be a potential claimant, if compensation for partial disability were permitted under the statute. The difficulty of procuring agreement among the members of the medical profession as to their determination of the existence or nonexistence of the disease in many given cases would probably result in an award of compensation to claimants when there was any evidence at all in the record to support an affirmative diagnosis of the existence of the disease. With increasing age employees show increased lung markings discernible by x-ray and it is probable that such markings would be the reason for medical diagnosis of the existence of the disease if the claimant had any silica exposure.
>
> "3. Claimant is not actually disabled unless the disease becomes complicated with an active infection resulting in total disability. When we consider

the definition of the term 'disability' as above related, contemplating some wage loss or loss of wage earning capacity, it does not seem inequitable to deny to the silicotic compensation for partial disability.

"*Second. Reasons for Granting Compensation:*

"1. The claimant has received some injury even though he is not disabled. Even though the claimant has not suffered wage loss he does have the disease which may later become complicated by other infection that would produce actual disability within the meaning of the term as used in the compensation statutes. When the disease becomes complicated with tuberculosis it is clear the claimant should receive the full benefits of the compensation act for permanent total disability. Not only for his own protection but for the protection of his fellow workmen he should be removed from employment until his infection is arrested and further suitable employment may be found for him.

"2. An employee having silicosis may be unable to obtain other employment when he leaves his job. The fact that he has silicosis would probably exclude him from employment in the trades or occupations where he is most proficient. With the extensive use of pre-employment examinations, the silicotic is undoubtedly under a severe handicap in obtaining other employment.

"3. Compensation for partial disability from other diseases is allowed. Even though the disease may not be disabling it may be contended that it is unfair to deny the benefit of partial disability awards to this limited group of potential claimants. Furthermore, the granting of such compensation may avoid a decision under the statute by the administrative agency that the claimant is totally disabled. It is possible that in the case of uncomplicated silicosis medical testimony may be offered on behalf of the claimant that, having the disease, it is inadvisable for him to continue in his occupation where he may be subject to continuing exposure to silicon dioxide dust. Upon the strength of such testimony the administrative agency may find that he is, in fact, totally disabled even though he would actually be able to perform other work in other employment where he is not exposed to the hazard of dust and may not suffer wage loss from such change of employment.

"*Conclusion. The following general conclusions may be stated:*

"1. In the absence of an accepted method for the medical evaluation of partial disability, legislative provisions denying compensation for partial disability seem proper and desirable. Further research is necessary and will be made to perfect some method for evaluating partial disability. If and when that method has the unqualified endorsement of the medical profession it would be most persuasive with legislators to effect the necessary or desired changes in our compensation laws.

"2. Whenever compensation for partial disability is permitted, there should be a limitation of the employer's liability so that it may be terminated at a fixed and determinable time. Unless this is done insurance carriers will continue to seek to avoid risks in industries where silica dust is a hazard because their liability cannot be determined.

"3. In the event an employee has silicosis and no disability within the meaning of that term as used in our compensation statutes it would seem to be desirable to afford to him the benefit of some compensation payment for the injury he has sustained so that if he cares to do so he may terminate his employment and seek employment in some other industry where he is not subject to the continuing hazard of silica dust exposure. A method similar to that provided under the statutes of North Carolina, West Virginia or Wisconsin would serve this prupose. That method would avoid the imposition upon industry of unjust and unreasonable compensation costs but give to the employee monetary benefit during the period of his adjustment in other employment."

The medical aspects of compensation for partial disability from silicosis were presented by Wright,[11] of the Truedeau Foundation. In part, he stated:

"The feasibility of adequately administering partial disability legislation in the silicotic will depend in a large measure upon the methods available to the physician by which he may assess the degree and cause of the disability. Hence a clarification of the limitations of these methods is essential. Since partial disability represents a deviation from normal ability, any evaluating method will be limited in utility by its capacity to correctly estimate normality. The meaning of the term 'normal' is familiar. It should be recognized, however, that although a method of measurement may be adequate, the deviations from the mean of a series of observations may be so large and so distributed that normality ceases to have a very significant meaning. There are four methods that readily suggest themselves as means of estimating disability.

"Some place great emphasis upon the roentgenogram in determining the presence or absence of disability. Whereas the x-ray is the cornerstone of diagnosing silicosis, experience seems to show that disability can be determined only in the most gross manner by this method. Our studies have convinced us that the roentgenogram is virtually useless in determining total disability, and is actually a pitfall of which one must be wary in determining lesser grades of disability. This view supports the general consensus.

"It has been suggested that daily 'work output' or 'earning power' be used as the measuring device of disability. The daily production of an individual depends upon personal incentive, natural physical and mental endowments and the nature of the job in relation to the output of the entire producing unit. Variations in these factors are responsible for the fluctuations of work output to be observed in a group of normal men and even in the same man from period to period. Moreover, these same factors control the ease with which one accomplishes his daily stint—some finishing ahead of time and others working hard to finish within the allotted period. It is apparent that one originally possessing a larger than needed capacity for work could suffer some degree of disability and still remain producing the usual output by simply woking more steadily throughout a given period. In fact, this is the mechanism of compensating for the disability due to aging. The precise limits of the range of this type of compensation are unknown, but they appear too large to make 'work output' a useful method of assessing disability. Moreover, since incentive plays so large a role in the daily output and the ease with which it is done, it would be very difficult to ascribe all variations in output to silicosis even though the latter be present. Although the output method will reveal disability, particularly when the work habits of the man are known, it would be difficult to consider this method trustworthy in all cases and it is probably of limited value in any.

"The third method consists of a careful physical examination and history together with such routine physiological tests as the electrocardiogram, the response of heart rate and breathing during mild exercise, sedimentation rate, sputum and urine examinations. Vital capacity, breath-holding time and the pressure developed by blowing against resistance are sometimes included.

"The scope of this presentation will not permit of an exhaustive analysis of the significance of the findings obtained by this approach. Those familiar with the art of medicine recognize the possibilities for error inherent in these procedures, even though extreme care to avoid them is observed. The problems of interpreting sign and symptom by both the examiner and the patient make this method a difficult one. Again, the large variation in the so-called normal man militates against the significance of any observation until the difference is marked. Moreover, examining physicians are usually required to give a decision concerning disability without the important advantage of observing the patient over a period of months. During one examination it is almost impossible to thoroughly grasp the complete situation in any given case. This works hardship enough in a decision confined to the establishment of total disability and would make the determination of partial disability particularly difficult. The Ontario Board of Referees have often found it necessary to repeatedly examine the claimant over a period of months in order to reach a clear decision. This method, when used by a physician whose knowledge of the patient extends through the years, thus affording him a basis for comparison and a thorough understanding of the complainant's psychological attitude and background, would be less liable to lead to error than any method now commonly used, although, even in the hands of very able clinicians, it leaves much to be desired.

"It seems highly desirable to attempt to measure body function in an objective manner in order to avoid the personal factor always present in clinical impressions. With this in view, a fourth approach (in reality merely an extension of the third) is to subject the claimant to rather complicated measurements designed to objectively test as fully as possible the capacity of the man as a unit to accomplish physical labor and to determine as specifically as possible the effect which the silicotic process has exerted upon pulmonary and cardiovascular function.

"The methods now used in such studies are not entirely satisfactory. The data obtained are still of an experimental nature, the interpretation of which is subject to change in view of later developments. However, such measurements now show conclusively that among normal men the individual will vary as much as 15-20 per cent above and below the mean for the group, though the method produces duplicate results within 5 per cent for the individual. In other words, an individual might be disabled in a given function by as much as 30-40 per cent and still be classed as a low normal. It is apparent that these tests would be extremely useful if some means were known whereby one could determine what the normal of any observation should be for the individual. Regardless of the imperfections now present in this approach, the data obtainable by these methods afford extremely valuable assistance to the evaluation of disability and merit further study.

"When disability has been shown to exist, it remains to be proven that silicosis is the causal agent. Our studies have amply confirmed the conclusions of others that simple discrete nodulation due to silicosis can exist without any evidence of disability. In fact, some cases of conglomerate silicosis fail to reveal demonstrable evidence of disability. One must recognize, therefore, that the presence of silicosis in a subject evidencing disability may be entirely coincidental. Disease of the cardiovascular system, of the kidneys, of the lungs, viz.: essential emphysema and asthma, may be coexistent and the actual causal agent of the disability. The evaluation of renal or cardiac function is most difficult, the more especially when complete failure has still not occurred. For this reason, it is often impossible to ascribe even total disability to coexistent silicosis—in regard to partial disability this problem is greatly magnified."

Various functional tests combined with methods of measurement have been designed to estimate the degree of disability in the silicotic. A description of the test suggested by Mayer was given in the author's previous textbook, *Occupational Diseases*. Blaisdell,[12] utilizing the tests developed by McCann, recommends the procedure to be described. To be of value to compensation officials the test would have to be made in a laboratory by those capable of fulfilling the requirements involved.

"The first part of the test is the measurement of the total pulmonary capacity and its various components. The measurement of the reserve air, tidal air, and complemental air is a simple procedure. The difficulty is in measuring the residual air. We use a modification of Christy's method, which is oxygen in dilution, without forced breathing. Of importance is not only the actual values of these subdivisions but their relation to one another. The measurement of the residual air is carried out in a special spirometer which will hold six or seven liters. The reserve air and complemental air can also be measured in this spirometer and the vital capacity in the small Collins spirometer. The nitrogen determination is carried out in duplicate by the use of advanced manometric methods. It is possible by roentgen ray measurements of the chest and certain regression formulas to determine what the total capacity should be and what these various partitions should be, provided no disease exists in the lungs. Six-foot plates are taken of the subject lying in the prone position, at maximum inspiration and at maximum expiration. These roentgenograms are measured. The area of the lung field is measured by means of a chronometer and recorded in square centimeters. We also measure the anteroposterior diameter of the chest at maximum inspiration. By multiplying the area of the lung field by the anteroposterior diameter of the chest, we arrive at a figure which is termed radiologic chest volume. From that we can calculate what the total pulmonary capacity should be and what the subdivisions of total capacity should be. We can predict from our roentgen ray measurements total capacity of the chest by this formula, the vital capacity by this formula, the residual air by this and the midcapacity by this. These are the predicted values for total capacity and subdivisions. The whole point of this is in a comparison of the predicted values with the values actually observed or measured. The predicted values will be extremely close to the observed values in a healthy subject. When disease occurs there is a greater and greater discrepancy between the predicted and the observed values.

"The second part of the test is a study of pulmonary ventilation before, during, and after exercise. For this purpose we have constructed a brake bicycle ergometer by the use of which we can give the subject any predetermined amount of exercise. The subject is connected to a series of Douglas bags. The volume of air contained in each bag is measured in the spirometer. We investigated a number of young, healthy subjects by this test, first at 300 kilogrammeters per minute. We computed the total ventilation at 300 kilogrammeters of exercise for a five minute period, which is 89.4 liters, which is very low. People suffering from pulmonary disease usually ventilate a great deal more than that. The healthy person increases the ventilatory activity by increasing his tidal volume, by increasing his respiratory rate. The respiratory rate in healthy subjects under exercise remains comparatively unchanged or increases but slightly. The total ventilation was only 133 liters; 183 at exercise and rest. The record of a man aged 38 showed that the vital capacity over total capacity was 69 per cent, which is good, the normal being about 75. This gives a picture of a normal person. He expands his chest 3 inches. At exercise his respiratory rate does not go over 20 per minute. It returns to normal quickly and his tidal volume is good. At 600 kilogrammeters of exercise we get a result that is similar. The remarkable thing about it is that this man has an advanced silicotic lesion, in all likelihood complicated by tuberculous infection, and yet shows no evidence of incapacity; he does not complain of inability to carry on his work.

"A man aged 50 does not show radiographically nearly the extensive silicotic process of the first patient. As the disease advances, his residual air has tremendously increased. His residual air is over 600 liters and is as great as his total predicted pulmonary capacity. His reserve air is greatly diminished and his vital capacity greatly diminished. His vital capacity makes up only 20 per cent of his total capacity. His residual air makes up almost 80 per cent of his total capacity, and his midcapacity about 85 per cent of his total capacity."

**Employability of the Person With Silicosis.**—Failure to appreciate the physical status of the person with silicosis by compensation boards and employers, as well as by the legal and medical professions, has caused considerable injustice. Men with varying degrees of fibrosis have been dismissed from their employment or refused other employment, although they did not evidence any signs of infection or disability. Yet it is a fact that men who have worked under silica hazard for more than thirty years and show evidence of marked fibrosis of the lungs are still able to work efficiently at hard manual labor. These men should be allowed to continue at their jobs. It must be remembered that silicosis does not disappear upon cessation of exposure. Discharge from employment will not alter or arrest the process. Only when there is evidence of disability or infection these individuals should be removed from their work. An exception to this rule is the new employee who reveals a too rapid development of the disease.

It may even be questioned whether those with massive conglomerate silicosis, who are short of breath and show signs of emphysema and chronic bronchitis, should be placed at rest. Many of these individuals have learned to accommodate themselves and prefer to continue a job which they have held so long. Gardner,[3] recognizing that many, and perhaps most, massive conglomerate lesions develop on a tuberculous basis, stated that "on first thought one is tempted to conclude that the subject should be given the benefit of sanatorium treatment, or at least be hospitalized to determine whether his infection is active. Experience in this country and elsewhere has demonstrated the fallacy of that procedure. These men have no symptoms attributable to infection; they resent such treatment and will not cooperate. Deprived of exercise their dyspnea becomes worse—perhaps because of increasing emphysema. No amount of rest will result in a decrease in size of their massive fibrotic areas. The expedient course is to allow them to work at jobs compatible with their abilities, so that they may retain their self respect and keep in reasonable physical condition."

Obviously, men in the class just mentioned should be watched carefully for the advent of tuberculosis, at which time for the good of their associates they should be removed from their occupation.

**Treatment.**—There is no treatment for silicosis which can be accepted as curative. In recent years aluminum has been advanced both as a preventive measure and as a means of amelioration. But even the most enthusiastic advocates of aluminum do not hesitate to emphasize that there is no substitute for rigid engineering and medical control of the environment.

In 1932 Denny, Robson, and Irwin[13] inaugurated an experimental investigation to determine whether there did not exist some substance which would combat the action of silica in the lungs. Accepting the theory that silica exerts its injurious effect upon animal tissue through a slow transformation into silicic acid in the presence of body fluids, these authors assumed that if the solubility of the silicious material retained in the lung could be sufficiently reduced, the usual fibrotic response would be modified or would not occur. A search was therefore undertaken for some nontoxic compound that would fulfill this purpose. After investigating many substances, Denny and Robson discovered in 1936 that the presence of small amounts of aluminum powder almost completely inhibited the solubility of silicious material in vitro. An intensive study of the mechanism involved later showed that the reduction in solubility was due chiefly to a coating of the silica particle with a thin film of a gelatinous hydrated alumina which, on drying, formed the crystalline alpha aluminum monohydrate boehmite ($Al_2O_3H_2O$). The presence of this adsorbed layer on the surface of the quartz was indicated by staining it with aurin (ammonium salt of aurin tricarboxylic acid) and proved by Germer and Storks* by means of electron diffraction patterns.

Inhalation experiments with animals were at once begun, using pure quartz dust with and without aluminum powder. These observers showed that while typical silicosis was produced in about five months in the control animals receiving silicia dust alone, the addition of 1 per cent aluminum powder to inhaled silica dust completely prevented the occurrence of silicosis, even after exposure had been prolonged up to twenty-two months. It was further shown that the inhaled aluminum powder should be of a particle size below 5 microns and that it might be inhaled independently of the silica dust. The aluminum powder employed in these experiments was freshly prepared in a ball mill charged with small aluminum pellets. These mills were placed in the animal cages and the powder dispersed into the atmosphere of the cage by a steady current of air. The inhalation of large quantities of aluminum powder for prolonged periods produced no harmful effects in the experimental animals.

Because of the beneficial effects noted in the experimental animals, Crombie, Blaisdell, and MacPherson[14] set up a clinic at Timmens, Ontario, for the treatment of silicosis in human beings. In the selection of cases to be treated, only men who were still exposed to silica dust and were still employed in the mines were considered, and only those with uncomplicated silicosis with measurable pulmonary disability were chosen.

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* Germer, L. H., and Storks, K. H.: Identification of Aluminum Hydroxide Films of Importance in Silicosis Prevention, Indust. & Engin. Chem. (Anal. Ed.), 11: 583, 1939.

"Before treatment was instituted, each individual was carefully studied clinically and radiographically, and a large number of respiratory function tests were carried out. The latter included a study of total lung volume and pulmonary ratios as described by McCann and his co-workers. Pulmonary ventilation studies were also made, employing standard exercise tests on a calibrated bicycle ergometer.

"Selected cases were treated by the daily inhalation of fine aluminum powder, freshly ground from small aluminum pellets in a specially constructed mill. A number of these powder mills were employed at various mines and treatment was usually given just before or following a shift. Treatment, administered daily, was begun by five minutes' inhalation through the powder mill. This period was gradually increased to thirty minutes daily and was continued over periods in some cases of almost a year, a small number of men receiving approximately 300 treatments, the majority, however, receiving about 200. About 50 patients have received this form of therapy, 34 of whom have completed the course prescribed."

**Results of Treatment.**—The results of Crombie, Blaisdell, and MacPherson[14] are as follows:

"This investigation has shown conclusively that the administration of aluminum powder in the manner described is entirely harmless and has proved definitely beneficial in a number of cases under our observation. Out of 34 cases studied, 19, or 55 per cent, have shown clinical improvement, apparent chiefly in the lessening or disappearance of shortness of breath, cough, pain in the chest, and fatigue. A reduction in the incidence of colds and a gain in weight have also been observed in many of the cases. While 15 cases have remained stationary, it must be emphasized that they are no worse and their condition has not progressed, in spite of continuous employment in silica dust during the period of our investigation.

"Respiratory function tests, repeated at intervals of about every three months, have shown improvement in 12 or about one-third of the treated cases. While 22 remained stationary, they are no worse in spite of continued exposure to silica dust. A group of controls, untreated by aluminum, haye shown progression of their disease in 66 per cent of nine cases while under observation."

Prior to the work of Denny, Robson and Irwin,[13] the English investigator, Kettle, had shown that quartz particles could be neutralized by coating their surface with iron. It was Kettle's work which suggested to Gardner that further attempts should be made along similar lines, and his group at Saranac was conducting experiments with one of the hydroxides of aluminum at about the same time that Denny, Robson, and Irwin were making their studies, although independently and without the other group's knowledge. Gardner and co-workers[15] summarized their experiments by stating:

"(1) If the alumina and quartz are injected in any way, so that they reach the same phagocytic cells, silicosis does not follow; (2) injection of gelatinous alumina after the quartz causes resolution and healing of immature silicotic tissue reactions, but fibrous nodules do not disappear; (3) the alumina has no harmful effect on normal animals; and (4) susceptibility to tuberculous infection is not increased by small doses of alumina that are effective against quartz, but excessive doses decrease resistance to tuberculosis, because the defense mechanism of the phagocytes is blocked. Then began a series of experiments on dust inhalation by guinea pigs, following a similar procedure to that used by the Canadian experimenters, but using alumina powder instead of aluminum. The results of the authors are in good agreement with those in the Canadian experiments, wherever comparison can be made, except in two minor details. Several forms of alumina, specially prepared by the Aluminum Co. of America, were tried before one was found comparable with metallic aluminum in its protective action. It was prepared so as to have maximum chemical activity, and is probably composed largely of amorphous alumina, with small amounts of alpha monohydrate and calcite. Work with this preparation is still in progress, and results to date have shown it to be effective in preventing silicosis when the distribution of silica is limited to the lungs. The treatment also arrests the development of tuberculous infection. The authors have to date conducted only a few experiments on men, five silicotic iron miners, with aluminum powder, and two silicotic lead miners with alumina. The response was not so favorable as in the experiments of Crombie and others. The number is much too small to permit conclusions to be drawn, but possibly the iron has interfered. Among problems on which experiments are still in progress is the cause of the apparent prompt relief of symptoms from the treatment without any change in the x-ray pattern of the dust reaction. The authors conclude: 'It may be said that confirmed experimental evidence proves beyond question that aluminum and aluminum hydrate, probably in amorphous state, specifically inhibit fibrous reactions to quartz, and that their administration will prevent progression of silicotic lesions and cause retrogression in immature tissue responses. Inhalation is probably the only effective method of administration. In the doses recommended for human therapy (five to thirty minutes a day or less frequently) no harmful results are anticipated. Aluminum therapy should not be used to the exclusion of recognized methods of dust control. Aluminum hydroxide possesses certain theoretical advantages over metallic aluminum and deserves serious clinical trial.' "

As this book goes to press, the machines and appliances are controlled by patent and can be used only by licensees. This fact, plus the possibility that this type of therapy might be used indiscriminately by industry without proper selection of subjects, led the Council on Industrial Health and the Council on Pharmacy and Chemistry to delay their approval of this type of therapy. These two Councils of the American Medical Association issued the following statement (April, 1946):

"The dissemination of reports on the effects of aluminum powder in the prevention and treatment of silicosis has aroused widespread interest and a demand for an authoritative statement regarding its value. At the present time the following statement would seem to be warranted; it is subject, however, to such modification as future knowledge and experience may indicate.

"1. In experimental animals the *prophylactic* use of aluminum inhibits the toxic action of relatively pure quartz.

"2. In man, industrial dust exposures often involve mixtures of various minerals in addition to quartz and other environmental variables to which experimental animals are not subject. Therefore human silicosis usually develops more slowly and is often modified in type. Only prolonged unbiased observation, with adequate control cases, will demonstrate whether the prophylactic results obtained with animals are applicable to man.

"3. Animal experiments have demonstrated that administration of high concentrations of amorphous hydrated alumina unfavorably influences resistance to tuberculosis. While this result has not yet been reported for metallic aluminum, caution in the application of all aluminum therapy to human beings is recommended.

"4. The use of aluminum might appear as an easy short-cut to healthful working conditions, thus saving large expenditures for ventilation and other control methods. Actually there is no substitute for the accepted methods of dust control.

"5. If industry indiscriminately treats all employees with aluminum dust, there may be aggravation of tuberculosis or other pulmonary conditions.

"6. The *therapeutic* use of aluminum in man appears to relieve symptoms in a very small number of cases in which silicosis develops rapidly. Experience in some groups is more favorable than in others.

"7. In view of these considerations it is recommended that the general application of aluminum therapy in industry be delayed until adequately and impartially controlled clinical observation demonstrates its effectiveness in preventing or alleviating silicosis in man. In the meantime, there should be no slackening in the control measures that have been found effective in reducing the incidence of dust diseases in industry."

## Case Reports.—

CASE 1.—This case is being reported because of an unusual complication and the rareness of the patient's first complaint.

In May, 1945, B. H., a 64-year-old man, suffered a contusion of the right shoulder at which time an x-ray examination was made. Although this picture was taken primarily to determine the presence or absence of bone injury, his physician noted the lung pathology and advised the man to cease working in the mine. The patient refused, however, stating that he felt well and capable of continuing his work.

In December of 1945 difficulty in swallowing and soon thereafter pain on swallowing developed. This condition progressed until the patient was unable to eat solid food. He lost considerable weight which he attributed to his inability to eat solid food. He was referred to the writer in May of 1946.

In questioning the patient more closely he recalled that for at least six months before he developed the difficulty in swallowing, he had noticed that his voice was hoarse and throat always dry.

*Past Medical History.*—Essentially negative.

*Occupational History.*—This patient first began mining in 1906 in Butte, Montana, and continued in that occupation until 1922. From 1922 to 1930 he operated a small hotel. From 1930 until 1945 he engaged as a hard rock miner in Arizona, Nevada, and California. He stated that for the past four or five years a wet process was in operation at his place of work, but prior to that time most of the mines made little attempt to control the dust hazard.

*Physical Examination.*—Weight, 135 pounds. Usual average weight, 180 pounds. Temperature, 99.4° F.; pulse, 110; face, flushed. The voice was very weak, barely audible, and the patient evidenced pain as he attempted to talk. Chest examination revealed a marked emaciation of the interspaces with deep depressions of the supra and infra clavicular regions. There was flaring of the anterior lower ribs, some prominence of the sternum, and a moderate kyphosis of the dorsal spine. Auscultation revealed râles in both apices, with increased bronchial breath sounds as well as intensification, especially on the right, whenever he used the voice in a whisper.

For a throat examination he was sent to Dr. Simon Jesberg of Los Angeles, who reported "an extensive tuberculosis of the larynx and hypopharynx. This is a secondary condition of the tuberculosis which complicates his silicosis."

Laboratory examination revealed blood sedimentation rate to be 30 mm. in one hour; sputum, Tubercle bacilli, 2 to 4 per high-power field.

The x-rays (Fig. 41) revealed closely packed discrete nodulation throughout both lung fields with loss of fine detail of the lung structure. Also may be seen an unusual number of large conglomerate nodules in the middle and upper zones.

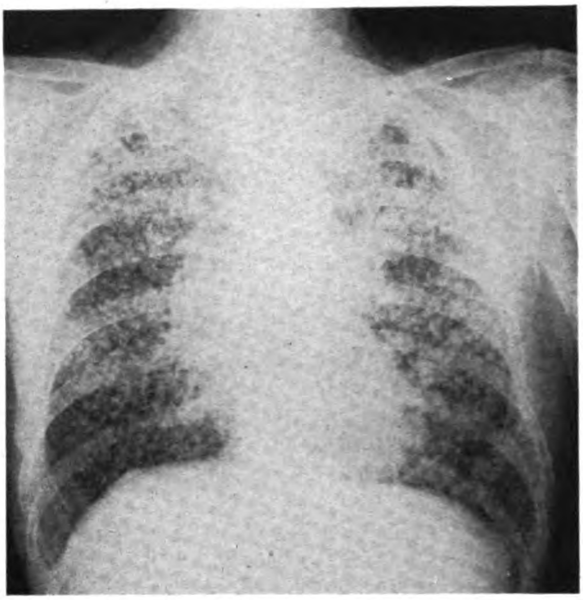

Fig. 41.—Far-advanced silicosis in a 63-year-old miner, showing widespread nodulation and areas of nodular conglomeration. Infection present (tuberculosis).

CASE 2.—The details of the clinical aspects of this case will not be given since they are not of essential interest. Suffice it to state that a male adult, who had been employed for a number of years, entered a claim for a permanent disability due to a "dust disease of the lungs." The physical examination did not indicate any disability of pulmonary origin. The x-rays revealed an old, healed, left apical tuberculosis but no evidence of pneumoconiosis.

It was recognized that the process in which the claimant was engaged involved the grinding of materials which did contain silicates, but it was also believed that there existed no exposure to free silicia. The operation consisted of grinding the barytes obtained from Battle Mountain, Nevada, and bentonite procured from a district east of Daggett, California. To the writer's knowledge, these substances had never before been *on trial* as a cause of silicosis. In the years of its existence the defendant company had not been confronted with a similar claim.

**Chemistry of Minerals.**—A chemical analysis of each is given in Table XVIII.

TABLE XVIII. TYPICAL ANALYSIS OF BARTITE GROUND AT LOS ANGELES

| | PERCENTAGE BY WEIGHT | | PERCENTAGE BY WEIGHT |
|---|---|---|---|
| $BaSO_4$ | 91.50 | CaO | Trace |
| $BaCO_3$ | 0.50 | Moisture | 0.10 |
| $Fe_2O_3 + Al_2O_3$ | 0.75 | | |
| $SiO_2$ | 6.50 | | 99.35 |

TABLE XIX. TYPICAL ANALYSIS OF HECTOR MINE-RUN BENTONITE

| | PERCENTAGE BY WEIGHT | | PERCENTAGE BY WEIGHT |
|---|---|---|---|
| Silica ($SiO_2$) | 31.50 | Potassium oxide ($K_2O$) | 0.65 |
| Alumina ($Al_2O_3$) | 7.21 | Moisture at 105° C. | 2.93 |
| Iron oxide ($Fe_2O_3$) | 0.11 | Combined water and volatile | 4.07 |
| Titanium dioxide ($TiO_2$) | 0.12 | Carbon dioxide ($CO_2$) | 16.42 |
| Calcium oxide (CaO) | 21.42 | Sulfuric anhydride ($SO_3$) | Trace |
| Magnesium oxide (MgO) | 9.05 | | |
| Sodium oxide ($Na_2O$) | 6.36 | | 99.84 |

Barytosis is chiefly mentioned in the foreign literature. Fifty cases of pneumoconiosis among workers in the barite mills in Italy were reported in which the pneumoconiosis presumably did not tend to cause tuberculosis but rather acute forms of lung affection. The American literature is practically devoid of any mention of barytosis, although Lanza produces an x-ray film showing diffuse, discrete nodulation in a person who worked in barium rock. In view of this negligible aid from the literature and because the defendant desired to have some concrete evidence against future claims, it was decided to seek all aid available. Dust counts had already established the fact that the atmosphere in which the claimant worked was one in which the concentration of dust was well within permissible limits.

*X-Ray Diffraction Analysis.*—It was decided to submit samples of the bentonite and barytes to Ernst A. Hauser, of the Massachusetts Institute of Technology, for x-ray diffraction analysis. These samples were: (1) composite Hector mill-run bentonite; (2) composite El Portal mill-run barytes; (3) typical Battle Mountain, Oakland, grind barytes; and (for the purpose of comparison) (4) a quantity of Ottawa sand, which is quite pure quartz. To these, also for comparison, Dr. Hauser added a standard pure quartz from his own stock. At the end of his investigation, he reported no evidence of uncombined silica in the samples submitted except that the El Portal barytes "might be faintly contaminated with uncombined silica, but according to the pattern, the amount of free silica was not over one per cent" (Fig. 42). This report of Dr. Hauser's offered substantial evidence of the negligible amount of silica present in this grinding process.

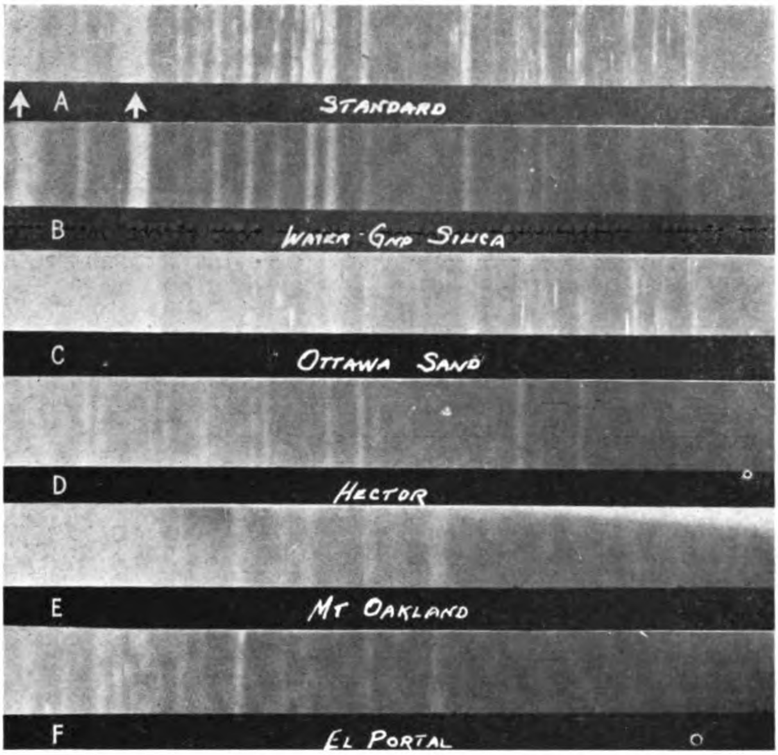

Fig. 42.—*A*, X-ray diffraction of sample quartz used for a standard. Arrows at left of film indicate the heavy silica line. *B*, A ground silica, about 2 μ in diameter, also used as a standard. *C*, Ottawa sand, a silica of average 30 μ diameter. *D*, California magnesium type bentonite. *E*, Nevada Battle Mountain barytes (Oakland grind), —325 mesh. *F*, California barytes, —325 mesh.

*X-Ray Diffraction Principle.*—The principle of the diffraction method is as follows:

When a beam of monochromatic x-rays is directed toward a finely divided crystalline substance, some of these x-rays are diffracted. If these diffracted x-rays come into contact with a photographic film, a definite pattern is produced and a reference point is formed by the undiffracted and unabsorbed x-rays that pass through the material. The diffraction pattern produced is governed by the nature and arrangement of the atoms in the crystalline material; that is, by the crystalline structure.

All evidence so far obtained indicates that no two crystalline substances give the same pattern, which, in a sense, is the "fingerprint" of the substance. The x-ray diffraction pattern, therefore, serves as a method of identifying crystalline substances.

According to the technique developed by Clark and Reynolds,* a pure crystalline powder, known not to be present in the dust being examined, is added to the unknown in a definite ratio and the diffraction pattern registered. The ratio of the density of a line sought to that of a near-by line of the added standard is determined protometrically. The ratio thus obtained is proportional to the line intensity of the substance sought, which in turn is proportional to the amount of the substance in the mixture. By reference to a curve that is prepared empirically from mixtures of known composition, the percentage of the constituent sought is obtainable at once. Fig. 43 shows the pattern for a typical dust sample compared with a standard pattern for ordinary quartz. This method, of course, is applicable to the determination of any selection of mineral constituents.

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* Clark, G. L., and Reynolds, D. H.: Quantitative Analysis of Mine Dusts; An X-ray Diffraction Method, Indust. & Engin. Chem. (Anal Ed.), 8: 36, 1936.

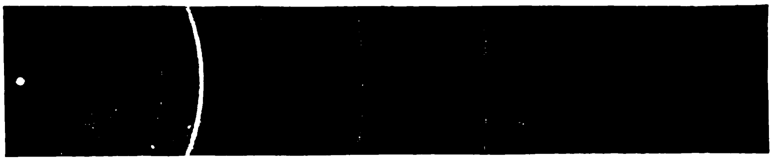

Fig. 43.—Powder diffraction pattern for quartz. (Clarke: Applied X-ray, New York, McGraw-Hill Book Co.)

It is to be appreciated by the reader that the problem in our case was to determine whether or not the claimant was subjected to a silicate-free silica mixture, and if free silica was present, to what extent. Some time after our investigation of this case, Louis H. Berkelhamer, of the Saranac Laboratory, reported additional applications of the x-ray diffraction method. He compares the values of the spectroscopic, chemical, petrographic, and x-ray diffraction methods of analysis in determining the presence of quartz in a given material.

According to Berkelhamer,* *spectroscopy* is of little value in this instance. Although it can be employed on very small samples, it demonstrates *only* the presence of the element silicon. By the unaided *chemical analysis* silicon dioxide is determined without the qualification as to the original state of the silica. Differential solution methods allow for an empirical estimate of quartz, but variations in particle size of the fine quartz fraction have so much influence upon its rate of solution that the results are not always reliable. A *petrographic* examination permits direct identification of quartz and, when combined with a chemical procedure, offers a satisfactory means of determining quartz. Petrographic methods, however, are difficult to apply to particles less than 5 microns in diameter, and it is particles 3 microns and under that are of the greatest pathologic significance. In this extremely small particle size range petrographic analysis must give way to *x-ray diffraction.*

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* Berkelhamer, L. H.: X-Ray Diffraction; an Important Tool in Pneumoconiosis Research, J. Indust. Hyg. & Toxicol., 23: 63, 1941.

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# CHAPTER XXIX

## ANTHRACOSILICOSIS

Most cases of silicosis result from exposure to an atmosphere which contains not pure silicia, but rather an admixture with other dusts. Anthracosilicosis is an example of this, and in this country it constitutes a disease confined chiefly to the miners in the hard coal district of eastern Pennsylvania.

**Findings in Governmental Survey.**—In May, 1933, at the request of the governor of Pennsylvania, the United States Public Health Service began a survey of the nature and prevalence of chronic incapacitating miner's asthma. During this study the occupational and medical histories and physical and roentgenologic examinations were recorded on 2,711 men. At the completion of this work, the following summary and findings were offered.[1]

*Definition.*—The term "anthracosilicosis" is a descriptive title for the form of pneumoconiosis commonly called "miner's asthma." It is a chronic disease due to breathing air containing dust generated in the various processes involved in the mining and preparation of anthracite coal. It is characterized anatomically by generalized fibrotic changes throughout both lungs with the presence of excessive amounts of carbonaceous and silicious material, usually by compensating emphysema, and often by cardiac changes in the later stages of the disease.

**Symptoms.**—The chief subjective symptoms found in the early stages were shortness of breath, cough, and pain in the chest; later, weakness and hemoptysis were frequently mentioned. The most common objective symptoms were dyspnea, prolonged expiration, decreased chest expansion, clubbing of the fingers, change in breath sounds, altered fremitus, and impaired resonance. In the more advanced stages (or in cases complicated by infection), persistent râles, cardiac impairment, loss of weight, and cyanosis were often seen, and usually there was more pulmonary infection and moderate or marked decreased capacity for work. Lung changes, general in distribution, often with more or less fixation of the diaphragm, were shown by fluoroscopic and x-ray-film examination.

**Basis of Diagnosis.**—Diagnosis was based on three important classes of findings: (a) histories, occupational and past medical; (b) clinical examination, symptoms, and physical findings; and (c) x-ray examination, fluoroscopic and film. For a definite diagnosis of anthracosilicosis, information from all three sources is required. In determining the role played by specific infection, laboratory examination should supplement the knowledge gained from the three sources mentioned. In certain borderline cases, repeated examinations may be necessary. A postmortem diagnosis should be based on the results of pathologic and chemical examination of the lungs. A complete diagnosis of anthracosilicosis includes a statement of any associated complications and of the extent and cause of permanent decreased capacity for work, if such is shown.

Of the various contributory factors observed in the development, course, and final effect of the disease, pulmonary infection and cardiac impairment appeared to be the most important, especially in the advanced stages of the disease.

**Groups Studied.**—These conclusions in regard to the nature and development of the disease were based on the examination of 2,711 men (about 96 per cent of the number on the payroll of three representative anthracite-coal-mining companies studied), on the findings for 135 disabled ex-miners who were hospitalized for observation, and on a smaller group examined in a state tuberculosis sanitarium.

The coal-mining employees were grouped occupationally, largely in accordance with the proportion of free silica found in the dust in which they were exposed.

No cases of anthracosilicosis were found in a control group composed of hard-coal-mining employees whose dust exposure averaged less than 5,000,000 particles per cubic foot of air.

**Prevalence and Exposure.**—The prevalence of anthracosilicosis among the entire group of employees was found to be about 23 per cent.

Among all except rock workers, less than 2 per cent of the men developed anthracosilicosis when the duration of employment was less than fifteen years, regardless of the amount of dust in the air.

Among men exposed from fifteen to twenty-four years to dust containing less than 5 per cent free silica, 14 per cent of those who had worked where the average dust count was from 100,000,000 to 199,000,000 particles per cubic foot, 29 per cent of those exposed to from 200,000,000 to 299,000,000 particles, and 58 per cent of the men who had worked for this period in more than 300,000,000 particles per cubic foot, developed anthracosilicosis.

Among men employed for more than twenty-five years in dust containing less than 5 per cent free silica, the proportion of persons found with anthracosilicosis under different concentrations of dust was as follows: from 5,000,000 to 99,000,000 particles, 7 per cent; from 100,000,000 to 199,000,000 particles, 54 per cent; from 200,000,000 to 299,000,000 particles, 71 per cent; 300,000,000 or more particles per cubic foot, 89 per cent.

With the exception of miners, their helpers, and rock workers, about 25 per cent of all the men employed underground developed anthracosilicosis after a working period of more than twenty-five years. This group was exposed to dust having a quartz content of about 13 per cent.

The prevalence of anthracosilicosis among persons who had been exposed for more than two or three years to dust, of which about 35 per cent was free silica, varied from 10 per cent among those who had worked in concentrations of less than 200,000,000 particles per cubic foot for less than fifteen years to 92 per cent among those who had been employed for more than twenty-five years in dust concentrations exceeding 200,000,000 particles per cubic foot, more than two or three years of which time was spent in rock work.

Age per se appeared to play a minor role in the development of anthracosilicosis.

**Safe Limits of Exposure.**—Analysis of the data for the purpose of determining safe limits of dust exposure indicated that employment in an atmosphere containing less than 50,000,000 dust particles per cubic foot would produce a negligible number of cases of anthracosilicosis when the quartz content of the dust was less than 5 per cent. In the gangways where the silica content of the dust was about 13 per cent, a safe limit appeared to be from 10,000,000 to 15,000,000 particles per cubic foot. The limit of toleration for rock workers was set tentatively at from 5,000,000 to 10,000,000 paricles per cubic foot of air.

**Respiratory Disease.**—Pulmonary infection increased with length of service more rapidly among the men in the haulageways than in the control group, and much more rapidly among the regular miners. The highest rates of pulmonary infection, however, were found among the rock workers of more than fifteen years' service.

The prevalence of pulmonary tuberculosis among the hard-coal-mining employees at ages below 35 years was slightly less than that found through studies of tuberculosis among male adults in the general population of the country. In the age group 35 to 44 years, however, the prevalence of tuberculosis was about twice the rate found in the general populations; at ages 45 to 54, about five times; and for the ages above 55, it was about ten times.

The highest prevalence of clinical pulmonary tuberculosis occurred among the rock workers (men who had been employed in rock loading or rock extraction for more than two or three years). After twenty years' service, of which more than two or three were in rock work, 37 per cent presented evidence of pulmonary tuberculosis.

In a group of 135 completely disabled former anthracite workers, which did not include any known cases of tuberculosis, 10 per cent proved positive for pulmonary tuberculosis.

Pulmonary infection (tuberculous and nontuberculous) was found among 58 per cent of the employed men having early anthracosilicosis and in 92 per cent of the workers in the more advanced stages.

Clinical pulmonary tuberculosis was diagnosed in 15 per cent of those with early anthracosilicosis and in 43 per cent of those in the more advanced stages.

In all groups combined, with the exception of the control group, about 20 per cent of the nontuberculous workers were diagnosed as having some respiratory disease other than tuberculosis. In the control group only 6 per cent had nontuberculous respiratory disease.

In the control group less than 2 per cent were found with moderate or marked physical impairment, causing decreased capacity for work, as compared with about 10 per cent among the regular miners and with approximately 13 per cent among the rock workers. With the exception of the rock workers, no group showed moderate or marked physical impairment in excess of that found among the controls when the period of employment was less than twenty years. However, an excess in the prevalence of slight impairment was found among the regular miners and among others exposed to dust containing less than 5 per cent free silica when they had worked from ten to twenty years in atmospheres containing more than 100,000,000 particles per cubic foot.

The correlations between exposure to dust and the evidence of constitutional changes left little doubt as to the etiological significance of the dust in the air breathed. Like correlations were found between the silica exposure and the extent of pulmonary changes.

Mortality from respiratory diseases was found to be much greater among anthracite workers than in the general adult male population of the country. The data indicated that underground work in the absence of dust did not predispose to fatal attacks of respiratory disease.

**Signs and Symptoms.**—As in silicosis, the first symptom is dyspnea. Initially this dyspnea does not require the patients to be in bed, but usually at the time they become short of breath on mild effort, their appearance takes on certain changes, there is a beginning loss of weight, the skin becomes pale or dusky, muscle weakness is noted, and the patient appears asthenic. A cough is common and productive of a blackish sputum. Because of the pleural adhesions and the fibrous changes causing fixation of the diaphragm or distortion of the mediastinum, pain is a frequent companion to the cough. Pain appears to be more common and seems to occur earlier than in silicosis with infection. Percussion note is impaired and breath sounds may be decreased or show signs of consolidation, depending upon underlying pathologic conditions.

**Pathology.**—

GROSS APPEARANCE.—The lungs are dark gray and firm and feel heavy. Black linear markings may be noted on the surfaces of the lungs. Adhesions between the parietal and visceral pleura are frequent. When sectioned these black lines are seen to run through the lungs, and there are also noted the disseminated, black nodules. In severe states, the pleural thickening and adhesions, as well as the fibrosis of the lung proper, tend to distort the shape of the lung. Coalescence of the nodules takes place in this stage, and emphysematous blebs become larger. Consolidation of those areas occurs usually subapically replacing normal tissue (Figs. 44 to 48).

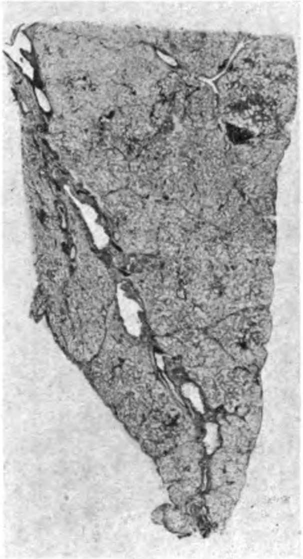

Fig. 44.

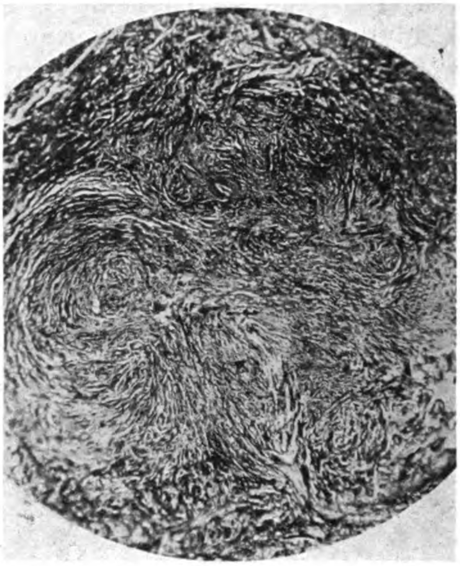

Fig. 45.

Fig. 44.—Simple pigmentation of uncomplicated anthracosis. The lungs appear almost normal. (Gardner.)

Fig. 45.—Details of an anthracosilicotic nodule. (Gardner.)

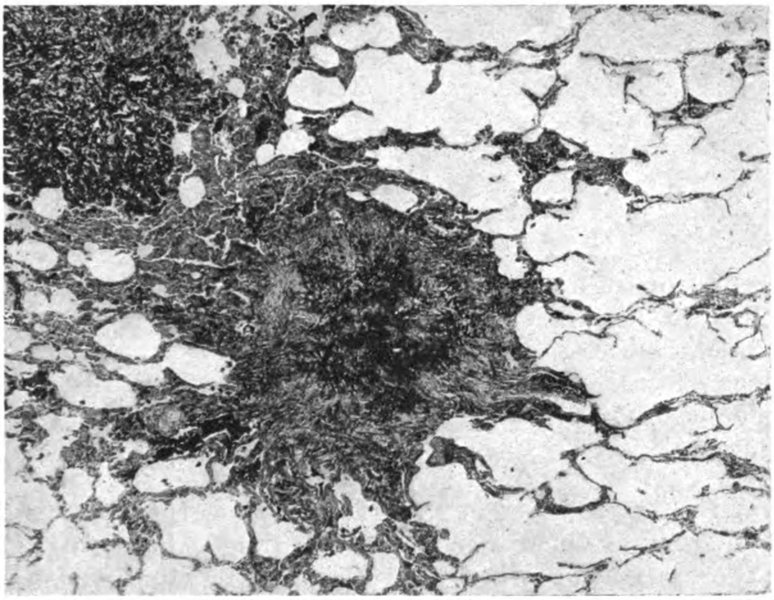

Fig. 46.—Details of an anthracosilicotic nodule with increased pigmentation. (Gardner.)

MICROSCOPIC APPEARANCE.—According to Dreesen and Jones,[2] black particles are present in the macrophages, but they are rarely seen free in the alveolar epithelial cells. Collections of "dust cells" are seen, however, in the perivascular and peribronchial lymphatics and adjacent interstitial tissue. Fibrous hyperplasia is observed quite early along the lymph channels and increases in certain areas to the formation of nodules. In the center of the smaller nodules, evidence of devascularization may be seen. The centers of larger nodules are, for the most part, well-formed, white, fibrous connective tissue and commonly reveal evidence of hyaline degeneration but are often free of appreciable amounts of carbon. In the periphery of the nodules, dust-laden macrophages are present. Free extracellular dust is noted in large quantities in the adjacent underlying dense connective tissue.

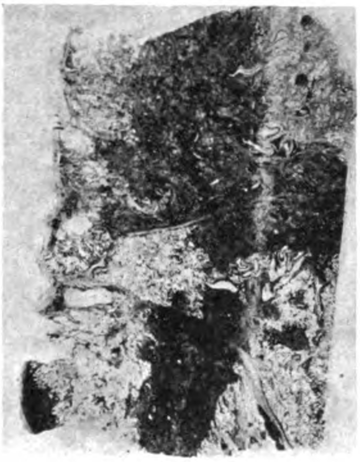

Fig. 47.

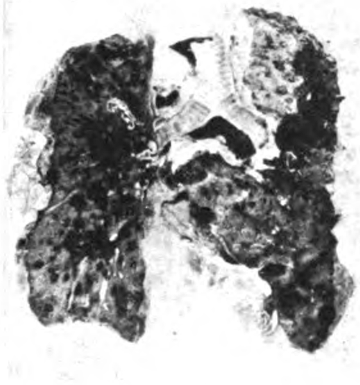

Fig. 48.

Fig. 47.—Conglomerate anthracosilicosis. (Gardner.)
Fig. 48.—Conglomerate anthracosilicosis. (Gardner.)

As confluence of the nodules occurs, the peripheral coal dust deposits are enclosed in the larger nodule formed. Areas of central, amorphous, finely granular necrosis are often seen. Fibrous hyperplasia and dense depositions of coal dust are seen in the peritracheal and pulmonary lymph glands, often to such an extent that histology of the organ is completely obscured.

**X-Ray Findings.**—The first typical appearance is that of an exaggeration of the normal linear markings of the lungs. Following this there is some increase in the general fibrosis. With continued exposure the film takes on a "ground-glass" appearance, the linear markings tend to disappear, and the generalized fibrosis is increased. At about this time very fine nodules may be scattered throughout the film. Further progress results in increase in number and size of the nodules. These nodules become larger than those seen in miliary tuberculosis, and at this time also emphysematous blebs will be noted. Finally, conglomerate masses are much in evidence, the emphysema has increased, and the generalized fibrosis is marked (Fig. 49).

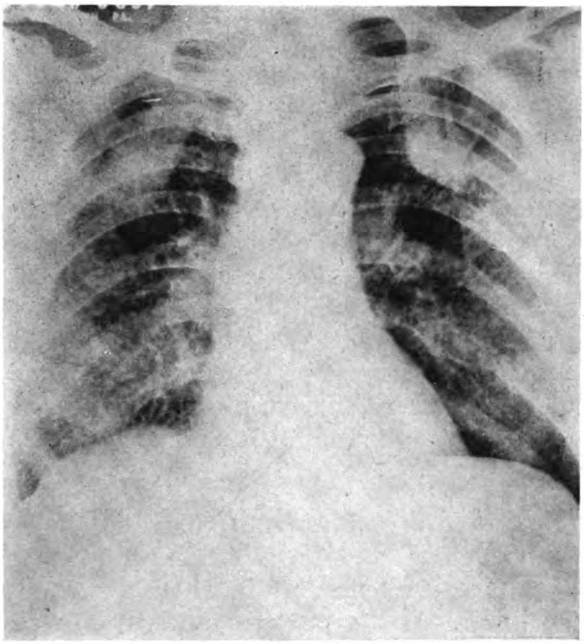

Fig. 49.—Anthracosilicosis proved by autopsy. (McCloskey.)

**Medicolegal Aspects.**—The determination of disability and the basis for such a determination correspond to those principles outlined under Silicosis.

## References

1. U. S. Public Health Service Bulletin 221, Dec., 1938.
2. Dreesen, W. C., and Jones, R. R.: Anthracosilicosis, J.A.M.A. **107**: 1179 (Oct. 10), 1936.

# CHAPTER XXX

# ASBESTOSIS

Asbestosis is a comparatively new disease. The first comprehensive reports regarding asbestosis came from the English writers in the years between 1920 and 1925. In America in the early thirties studies were made by Lanza, Sayers, Bloomfield, and others.

Asbestos is a hydrated magnesium silicate. Occupational exposure occurs in those trades where it is used for packing, insulating, or fireproofing, or where it is combined with cotton or other material in textile processes.

**Signs and Symptoms.**—As in silicosis,[1] the first sign is dyspnea, the cough is usually dry, and auscultation reveals few, if any, physical signs. Loss of weight is usually noticeable, the color is pale, and cyanosis is a fairly early sign. After some time, fine, crackling râles may be heard, and the chest appears emaciated and lacks the robust character noticed in silicosis. Early asbestosis must be differentiated from acute emphysema. If tuberculosis is present, the symptoms may take on the character of this disease, but this complication is far less frequent than in silicosis.

**Pathology.—**

PULMONARY CHANGES.—The pulmonary fibrosis of asbestosis is diffuse in character, peribronchial, and basal in location in contrast to silicosis in which the fibrosis is nodular in character and present in the upper part of the lungs. As the process continues, the fibrosis extends into almost all portions of the lung tissue. Bronchiolectasis and bronchiectasis are present within the substance of the fibroid areas, and bronchopneumonia and acute tracheobronchitis often cause the death of these patients. The pleurae, especially in the basal regions, are thickened and adherent. In some cases the pleural sac is completely obliterated; in others a fibrous or serous exudate may be present within it.

**Action of Asbestos and Silica.**—The difference between chrysotile asbestos and quartz silica in their mode of action has caused considerable speculation as well as experimentation. Gardner[2] particularly, at the Saranac Laboratory, carried on intensive study of these two materials. He felt that while the action of free silica is chemical in nature, the action of asbestos is mechanical. He stated:

"We have come to the conclusion that inhaled asbestos fibres are irritating not because they are silicates but because they are stiff fibres which mechanically irritate the lungs. Unlike the free silicas, these minerals will not stimulate fibroblasts in any part of the body; only those in the lungs are affected. It was inferred that these organs were affected because the movements of respiration are so much more rapid and continuous than those of other viscera. Then it was discovered that if asbestos was ground very finely so that few of the fibres were longer that 2 microns in length the irritating property of the asbestos was practically destroyed. Inhalation experiments with such fine chrysotile asbestos were carried out over a period of several years. No fibrosis has developed in spite of the fact that an average atmospheric concentration of 125 million particles per cubic feet of air has been maintained. In contrast in a previously reported experiment one-third this concentration of long fibre asbestos dust produced well marked fibrosis after about two years."

If the effect of asbestos were chemical, one would expect that a decrease in size would accelerate tissue response. With free silica large particles have little effect, but as their size decreases cellular reaction becomes more vigorous and even constitutional symptoms may ensue. With fibrous asbestos the reverse is true.

The histology of early asbestosis does not suggest a chemical injury. Even under the most extreme conditions that can be created by artificial injection there is no preliminary phase of tissue necrosis with infiltration of leucocytes as occurs with high concentrations of very fine quartz. The connective tissue cells merely multiply very slowly in areas where the asbestos fibres are caught in the bronchioles. As collagen forms and contracts, the air spaces are obliterated by scar tissue. In experimental animals, at least, this change is not a progressive one after cessation of exposure to the dust as is the case in the response to quartz. Perhaps the reason is the deposition of the peculiar iron-containing coating on the surface of the inhaled fibres giving rise to the characteristic "asbestosis bodies" (Figs. 50 and 51).

Finally, it is most suggestive that among dozens of different silicate minerals only the five known as asbestos, which are unique because they are fibrous in structure, should be commonly recognized as pulmonary irritants. The variation in chemical composition within this group is greater than that between them and many other silicates. In fact, chrysotile asbestos has the same chemical formula as a nonfibrous silicate, serpentine, which is physiologically inert. Obviously irritation would seem to be associated with the physical rather than the chemical composition of these minerals.

One point of practical significance may be indicated by these observations: namely, that very finely ground asbestos is not dangerous. This conclusion has support in clinical observation, for it has long been known that at the Thetford Mills there was no clinical asbestosis even though in former years the atmosphere was very dusty and the dust was extremely fine. The fact that fabrication of fibres of the same mineral in American plants could produce disease was one of the puzzling fea-

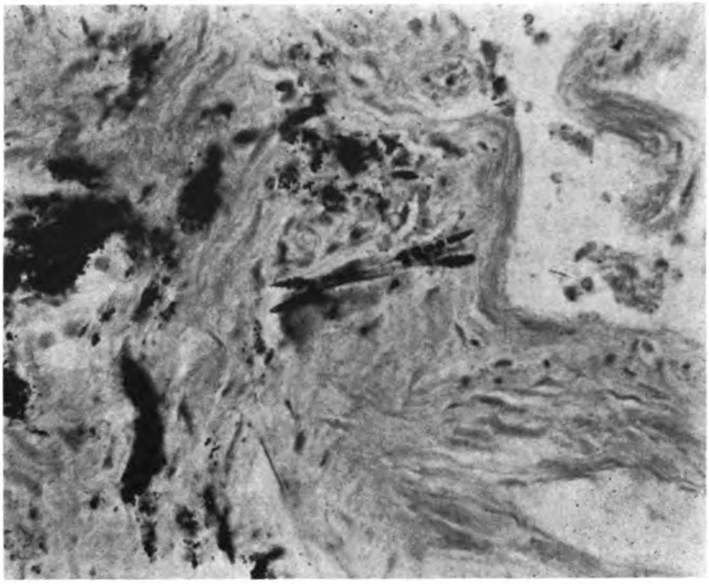

Fig. 50.—Right lung, showing markedly thickened pleura with fusion of interlobar pleura. Dense fibrous tags over entire surface. (U. S. Public Health Service, Bulletin 241.)

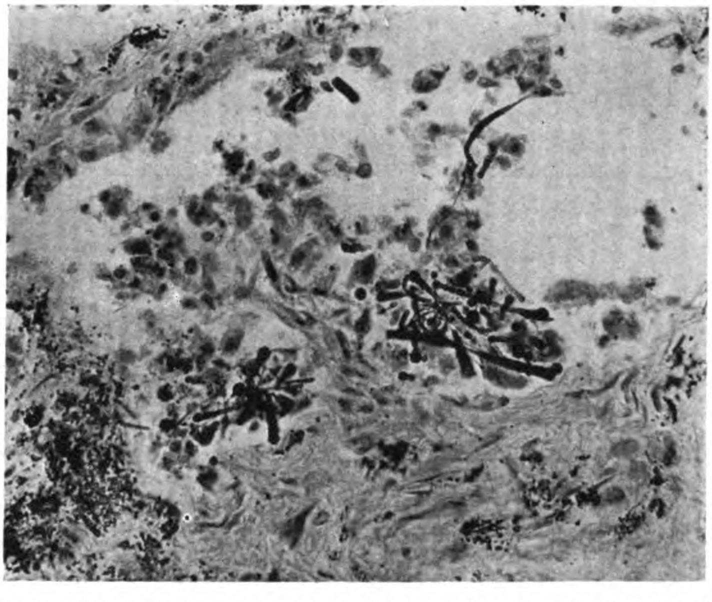

Fig. 51.—Asbestosis bodies accompanied by giant cells. (U. S. Army Medical Museum, Specimen 52255.)

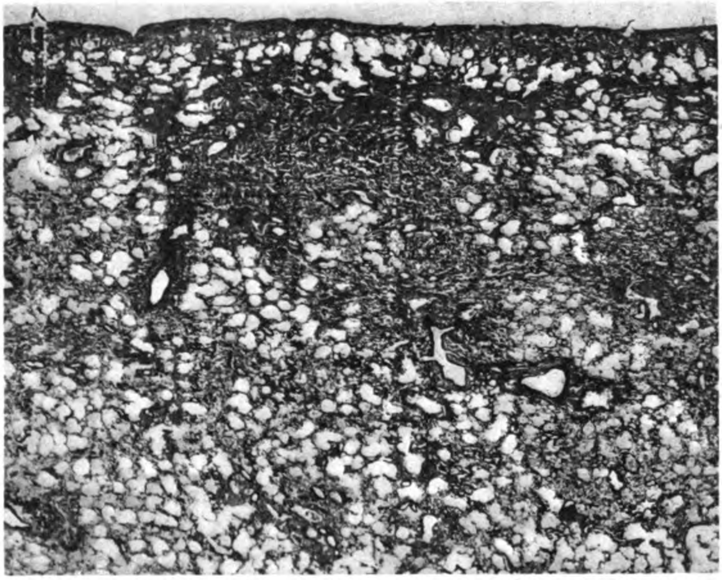

Fig. 52.—Section of a lung in asbestosis. (Gardner.)

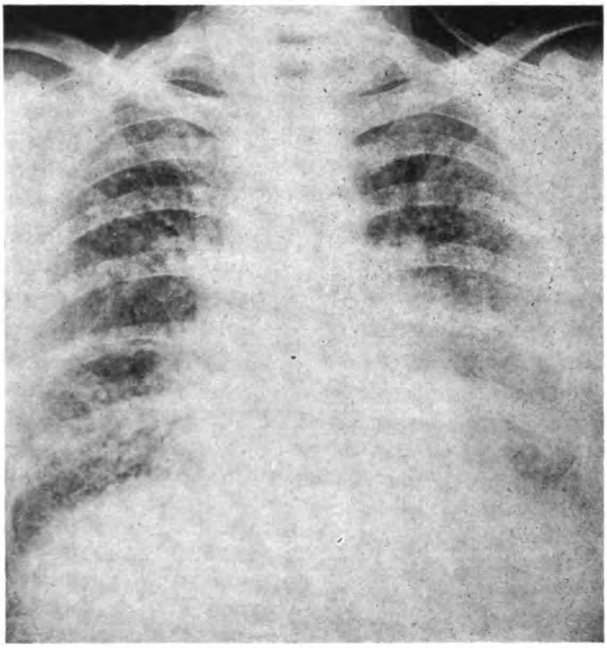

Fig. 53.—X-ray film of a patient with asbestosis. (Pendergrass.)

tures of this disease. But these experiments offer a plausible explanation. The fine dust in the mills is composed of serpentine and extremely short chrysotile fibres; that in the spinning and weaving mills contains many more long fibres.

**Microscopic Appearance.**—In the early phases of the disease there is a thickening of the alveolar septa as a result of fibroblastic proliferation. The alveolar spaces contain numerous alveolar phagocytes. With progression of the disease, fibrosis becomes more marked and the alveolar structure gradually disappears, and in its place there is now dense fibrous tissue. The few remaining alveoli which lie in the area of fibrous tissue are lined by a low cuboidal epithelium and have a glandular appearance (Fig. 52).

Scattered throughout the lung in both the diseased and healthy parts are spindle-shaped structures (first described by McDonald*) known as asbestosis bodies. These bodies are slender in their center and bulbous at each extremity. Many are arranged like strings of graduated beads with the largest at the end of the chain.

**X-Ray Findings.**—Certain peculiarities exist here as contrasted to silicosis. In asbestosis, the lesions may be bilateral or largely unilateral. According to the experience of Pendergrass,[3] the roentgenologic findings in asbestosis are largely limited to the lower half or two-thirds of the lung fields, while in silicosis the upper portions of the lungs are also involved. In moderately advanced asbestosis, x-rays will reveal lessened ventilation of the lung fields, the parietal pleura is thickened, and there is a "ground-glass" appearance to the picture. The vascular shadows lose their identity. Nodulation is absent. Pendergrass[3] feels that a roentgenologic diagnosis of early asbestosis is unreliable and that the condition must be moderately or markedly advanced to make a differential diagnosis from the roentgenograms (Fig. 53).

**Medicolegal Aspects.**—It is felt that the time necessary to develop asbestosis is, on the average, from seven to nine years in a fairly high concentration; with a less severe concentration, from fifteen to twenty years. The allowable concentration is 10,000,000 particles per cubic foot of air, of a size between 0.5 and 5.

Once established, the disease is progressive, even after the cessation of exposure. The basis for diagnosis is similar to that given under Silicosis.

### References

1. Lanza, A. J.: Asbestosis, J.A.M.A. **106**: 368 (Feb.), 1936.
2. Gardner, L. U.: Recent Developments in Relation to Silicosis, Indust. Med. **9**: 45 (Feb.), 1940.
3. Pendergrass, E. P.: Silicosis and Asbestosis, New York, 1938, Oxford University Press.

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* McDonald, S.: Histology of Pulmonary Asbestosis, Brit. M. J., **2**: 1025, 1927.

# CHAPTER XXXI

# SILICIOUS DUSTS OF UNDETERMINED PATHOLOGIC STATUS

It has long been the opinion of most investigators that only free (crystalline) silica and asbestos (a silicate) are capable of producing a disabling pulmonary fibrosis. For many years many of the silicates have been studied experimentally, and their action has been observed clinically with no evidence that these silicates are harmful to any appreciable degree. Recently, however, several of the silicates have been suspected of being capable of producing a fibrosis not unlike that due to free silica. As yet no conclusive opinions have been reached and until more investigation by capable authorities is carried out, the status of these dusts must remain undecided.

## Talc

Talc is a hydrous magnesium silicate. When it is mined, talc may be accompanied by other minerals such as serpentine, dolomite, and tremolite. Occupational exposure includes, in addition to mining the mineral, the preparation of talc for fillers in paint, linoleum, paper, dusting agents, and cosmetics.

In 1933 Dreessen[1] and subsequently in 1935 Dreessen and Dalla Valle[2] studied the effects of talc mining in Georgia and Vermont and noted that x-ray pictures revealed diffuse fibrosis, but whether this constituted a disabling pneumoconiosis was debatable. Porro and co-workers[3] took the definite position that talc did cause a disabling pulmonary fibrosis and attempted to correlate the mining and milling of talc with a morbidity among these workers. In 1942 they made the following observations on fifteen cases with a history of exposure to talc:

> "The symptoms are those characteristic of disabling pneumoconiosis: dyspnea, cough, chest pain and weakness. Vital capacity was extremely low. The gross pulmonary changes in all cases are stiffening of the lungs, poorly outlined grayish bands and nodules appearing in the parenchyma and moderate enlargement of the tracheobronchial lymph nodes. Emphysema is universally present but is not grossly recognized in the mild case. In all except the least involved case, extensive intrapleural adhesions are found and in all advanced cases there is a cor pulmonale. Microscopic changes consist chiefly in irregular areas and bands of cellular fibrosis surrounding the vessels and bronchi and producing thickening of the alveolar wall. Histiocytes and fibroblasts are numerous in proportion to collagen fibers. Asbestosis bodies are frequently present in the lesions. This implies a degree of similarity between asbestosis and pneumoconiosis due to talc. While tuberculosis or other complications may be present, pneumoconiosis in itself is a reaction of living tissue to injury and therefore an inflammatory disease resembling other chronic inflammation. Diffuse haziness is the most constant roentgen abnormality and is probably the

earliest detectable change. The general background of the pulmonary field may have a fine indefinite reticulation of lines. The appearance suggests that the haziness due to interstitial fibrosis has been modified by a fine emphysema. Another manifestation is usually general nodulation. Confluence of lesions and haziness usually begin in the areas of distribution of the lower lobe trunks. The diagnosis is made on the history of an adequate exposure and on the roentgen signs. If disability is present the characteristic symptoms are significant. If uncomplicated, the symptoms of talc pneumoconiosis are so unlike those of pulmonary tuberculosis that a differential diagnosis is comparatively easy. In uncomplicated talc pneumoconiosis the onset of symptoms marks the beginning of a disability from which recovery appears to be impossible. It is desirable to withdraw the worker from exposure prior to this time. There are roentgen changes before the onset of dyspnea or other subjective symptoms. The determination of vital capacity may be an effectual routine procedure in selecting workers that should be more thoroughly studied as potential subjects for withdrawal from further exposure. Standard exercise tests for the objective periodic determination of dyspnea may aid in the discovery of early pulmonary fibrosis."

In 1946, Porro and Levine[4] reported an autopsy examination of a talc worker, aged 45, who had been unable to work for three years because of difficulty in breathing. Two weeks before he had started to expectorate bloody sputum and to complain of pain in the left side of the chest. Roentgenologic observation revealed the left lung field below the level of the first interspace to be completely obscured and in the right lung field a dense reticulation. The dyspnea continued to be severe, and the patient died. The gross necroptic aspects as well as the microscopic changes were described. One section of the lung consisted largely of hemorrhagic infarct. Other sections revealed many alveoli containing fluid, erythrocytes, and macrophages containing hemosiderin. Some of the alveoli contained asbestos bodies. Localized emphysema and atelectasis were present. Many of the interlobular septa and some alveolar walls were thickened by a fibrocellular reaction consisting of histiocytes, scattered lymphocytes, fibroblasts, collagen fibers, and amorphous brown pigment. No tubercles or silicotic nodules were found.

In 1942, following the publication of the author's book on *Occupational Diseases*, a communication was received from Dr. Daniel F. Levy, of New Haven, Connecticut, in which he questioned the statement that talc produces no undue pulmonary fibrosis. In this communication (April 21, 1942) Levy stated:

"I have had occasion to be concerned rather recently with a case in which there was a long history of inhalation of soapstone or talc used for coating rubber wires. The patient developed fibrosis and tuberculosis and died. At autopsy (the findings of which I was permitted to observe) it was most interesting to observe that there was fibrosis, fibrous pleural adhesions, and fibroulcerative pulmonary tuberculosis. Microscopically, typical asbestosis bodies were seen in addition to other findings such as are present with the diagnoses just enumerated. This man had never been exposed to asbestos.

"In connection with the subject, I have examined a considerable amount of literature which would indicate that talc dust is not innocuous. I have in mind such matters as the report of Dreessen, W. C., and DallaValle, T. M.: Effects of Exposure to Dust in Two Georgia Talc Mills and Mines, Pub. Health Rep. **50:** 131, 1935. I should furthermore like to state that Dr. Zimmerman, pathologist at Yale University, has told me that he has met this same picture of asbestosis bodies following talc inhalation in other autopsies performed by him."

## Bauxite

Inhalation of aluminum powder or dust has been considered harmless to the lungs. In fact, as pointed out in the chapter on Silicosis, aluminum inhalations have been considered effective in the prevention of silicosis. Studies made of a large group of workers at the plant of the Aluminum Company of America at New Kensington, Pa., revealed that inhalation of aluminum powder even in large amounts and over a period of many years had caused no lung damage.

In the spring of 1946, while attending the annual meeting of the Ramazzinians, Dr. J. G. Cunningham, Director of Industrial Hygiene of the Department of Health of Ontario, Canada, told the writer of some unusual pulmonary disturbances that were occurring among the furnace workers processing bauxite. At that time, he and Dr. A. R. Riddell, his associate, were working on the problem. But before relating their subsequent report it is best that the composition of bauxite be explained.

Most of the aluminum found in the earth's crust is locked up in soluble crystalline silicates, such as feldspar and mica, which make up a large part of granite and other igneous rocks. As these rocks slowly weather and are leached by running water during the lapse of centuries, they may leave a residue of kaolin. Further weathering of kaolin gradually removes the silica, leaving behind bauxite, the chief ore of aluminum. Chemically it is hydrous aluminum oxide. It contains considerable amounts of soluble impurities such as silica, oxides of iron, and titanium. Emery is an anhydrous, impure, aluminum oxide. A purer form of aluminum oxide, more definite crystalline, is corundum.

When this book was nearing its final stages of preparation the author wrote to Dr. Cunningham and Dr. Riddell, seeking some information as to their conclusions regarding the cause of the illness existing among the bauxite furnace workers. In a letter of January 29, 1947, Dr. Riddell replied as follows:

"*History.*—The condition first came to attention in February, 1942, when a young man, aged 33, was referred for examination by Dr. C. G. Shaver, of the Niagara Peninsula Sanatorium, St. Catharines. Dr. Shaver noted the history of exposure, but thought little of it at the time. Two similar cases were seen by him in August, 1942. These men had worked in the same plant as the original case at the same type of work, and the clinical picture presented was so unusual that he felt he was dealing with a condition of occupational origin.

"Over some months several other cases were seen. Dr. Shaver appealed to this department. He stated that he was sure that he was dealing with an unusual disease of occupational origin. At first his contention was received with some scepticism but he finally convinced me, and I convinced Dr. Cunningham and the others concerned. An investigation into the process was commenced and is proceeding. An additional group of cases have come to our attention. We have obtained a number of autopsies which are quite convincing.

"The process concerned was carried on in four plants in the neighborhood of Niagara Falls, Ontario. It is the manufacture of corundum and artificial abrasives. In the process, bauxite, to which is added small quantities of iron and coal or coke, is subjected in metal pots to a temperature of approximately 2,000° C. The pots are approximately 6′ by 6′. Into these large carbon electrodes are lowered and the necessary heat is produced by the electric arc. The mixed material is added to the pots prior to the lowering of the electrodes and from time to time during the process. It is added to the pots by workmen who shovel the mixture.

"During the process dense white fumes are evolved. These are largely carried upward by the heat draft from the pots to openings in the roofs of the furnace rooms, where they escape. There is, however, considerable contamination of the atmosphere in the neighborhood of the furnace tenders. Crane operators and certain other workers also are exposed to large quantities of this fume.

"Analysis of the fume shows that it consists of extremely fine particles of alumina (Al. 203) and silica, the proportions being approximately alumina 60 per cent and silica 25 per cent. In addition to these substances small quantities of many other materials are present in the fumes.

"This process has been carried on for many years, but no cases such as those to be described had previously come to our attention, nor as far as can be determined had any similar case been seen by other persons. The processes were carefully canvassed to see whether any recent change in procedure or in materials had been instituted in any of the plants. No change had apparently been made, except that during the period when these cases occurred production was increased. This increase was in the dimensions of about ten times. There was no corresponding increase in the working staffs. Exposure of those working during the period of increased activity would therefore be materially greater than previously.

"An x-ray survey, supplemented by clinical examination when this was considered necessary, was made of the working staffs of the four plants involved. Some 1,913 workers were examined. Three hundred forty-four were furnace tenders or worked in the neighborhood of the furnaces. From this group of furnace men upwards of 35 persons have been found whose chest films show shadowing which we consider to be characteristic, and in addition there are a number of others with x-ray changes which we have labelled as doubtful. It is of interest that some of this doubtful group have progressed since they were first seen and we can now consider them definite. In one case in particular special comment might be made. A young man aged 32, who had been employed as a furnace tender for two years, joined the armed forces in September, 1943. He served in England and on the Western Front. He returned to Canada in the Fall of 1945 and was discharged in July, 1946. While on service he had what was diagnosed as pneumonitis in February, 1945. His case was investigated in a military hospital and was labelled as pulmonary tuberculosis. It is evident that the clinicians were somewhat puzzled because they had re-x-rayed him very frequently. While under observation he developed a spontaneous pneumothorax and was admitted to the Niagara Peninsula Sanatorium, under Dr. Shaver, at his request. On reviewing this man's enlistment film, it is evident that some slight changes are present. These were evidently not considered sufficient to bar him from military service. Films prepared during the period when he was under observation in the Military Hospital show quite characteristic shadowing.

"I know of no condition in which the chest film corresponds so closely to the changes found on pathologic examination. We, in our Survey, divided the cases into 'early' and 'well established.'

"In early cases an increase is seen in the subapical area in the upper lung fields. This is bilateral and the shadowing is of a somewhat lacelike or granular character. As the condition extends the shadowing becomes more widespread. In very marked cases practically the whole lung field may be involved.

"Advanced cases show less of the lacelike character in the shadowing. The appearance in these is somewhat like that seen in irregular types of silicotic fibrosis. In addition, however, the mediastinum is widened, sometimes markedly so, and the diaphragm is very markedly distorted. In contradistinction to what is seen in ordinary dust fibrosis, the general trunk and linear shadowing is not unduly prominent and the shadows of the hili are not marked. Spontaneous pneumothorax characterizes the more advanced cases. The collapse is often bilateral. Where unilateral collapse occurs there is a marked shift of the mediastinum away from the side of the collapse. Also, as a rule the diaphragm returns to its normal position following a collapse.

"*Symptoms.*—In early cases, symptoms are slight or entirely absent. Not uncommonly, those affected gave a history of some cough and expectoration over a period of time. The sputum was generally frothy in character. Not infrequently the case sought medical advice because of the sudden occurrence of severe dyspnea. Sometimes, but not invariably, this was associated with pain. Most of those seen complained of a tightness in the chest, which had been present for indefinite periods. Most of those with severe symptoms had lost considerable weight and many complained of loss of appetite and sleeplessness. The well-established cases all complained of moderate dyspnea on exertion. Of course, in severe cases this was very marked and sometimes it was present even while the patient was at rest.

"No special findings were elicited in clinical examination. As a rule, in the early cases, the examination was essentially negative. In the presence of spontaneous pneumothorax, hyper-resonance occurred with increasingly diminished breath sounds. In several cases adventitious sounds, rales, and rhonchi were present. Not infrequently, areas of impairment of resonance were made out. These often occurred toward the lung bases and in several instances could be ascribed to the marked elevation of the diaphragm on the side concerned.

"I mentioned that we have had upwards of 35 cases from an over-all population of some 344 persons in exposure. I have no doubt that this finding is on the low side. From among these there have been 7 deaths and at the present time at least another half dozen individuals with serious disability.

"This condition is receiving our concentrated attention. Investigations are proceeding at the plants from which these cases have come and also some work is in progress in the laboratory. We are not yet prepared to state with any assurance what the specific causative agent is, except insofar as to say that it is occasioned by inhalation of the furnace fume. Whether any specific part of the fume is particularly responsible we do not yet know.

"Incidentally these cases appear from the descriptions to be quite similar to certain cases which occurred in Germany during the war years. In Germany, these workers were exposed to the dust of aluminum which is apparently pure. The condition here occurred also during war years. Unfortunately, we have no back films on our cases, except in one instance. It may be significant that a film on one of these men which was prepared in 1936 shows some shadowing of the type which we associate with this disease. This man is not, however, seriously disabled.

"For the most part the cases occurred in persons who were exposed after 1939. Many of those seriously affected were young, 25 to 35 years of age. The shortest exposure was 22 months. We have had exposures of approximately 32 to 36 months."

In a separate communication Dr. Cunningham wrote: "I do not think that tuberculosis has been present in any of these cases. The x-rays present a wide shadow in the aortic area with an interlacing fibrosis in the upper zones. The picture presents a fibrosis which is not characteristic, that is, there is no whorl formation as encountered in silicosis."

The following series of films (Figs. 54 to 60) illustrate the case of L. R., a 43-year old bauxite worker, who was first seen on July 20, 1943, when he complained of loss of weight and difficulty in swallowing. An x-ray film taken on that date revealed a fine type of shadowing which is now considered as characteristic of the early stages of this condition.

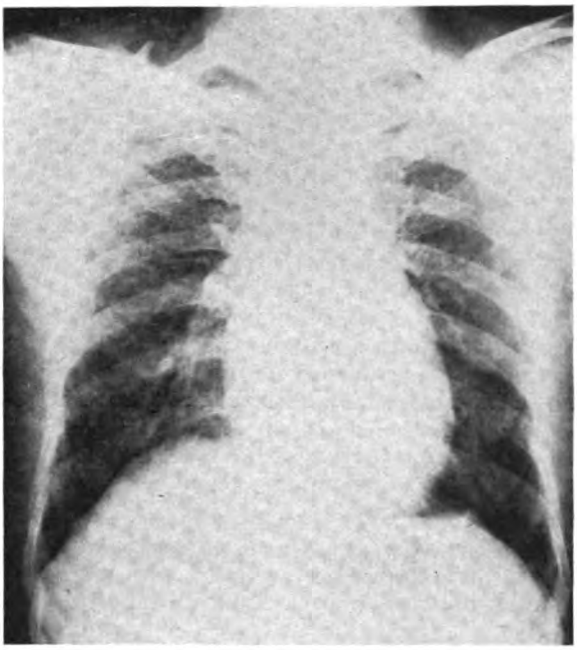

Fig. 54.—Film of bauxite worker taken July 20, 1943, shows "fine shadowing" of upper portion of lungs.

His previous occupation had been that of a blacksmith and also a construction worker. In June of 1941 he began his work in processing bauxite, being engaged as a furnace feeder, shoveling the mix into the electric furnace, until December, 1943. In December, 1943, he developed discomfort in the chest with considerable dyspnea. Early in 1944 he developed a sudden, severe pain in the right chest again associated with marked dyspnea.

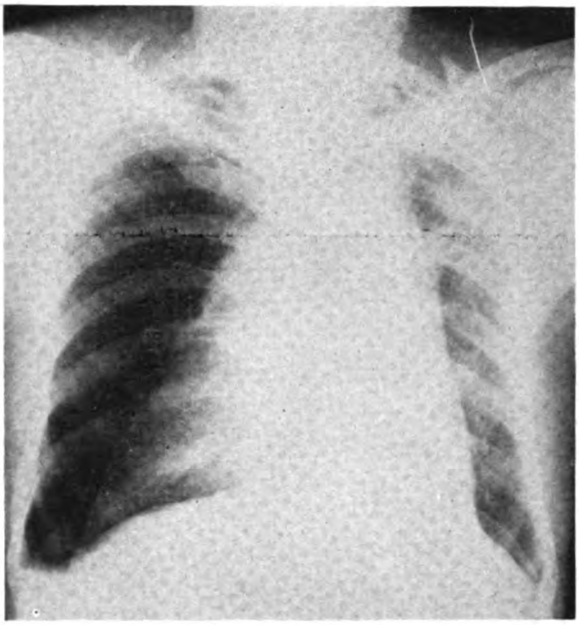

Fig. 55.—Film of worker shown in Fig. 54 taken Dec. 14, 1943, shows collapse of right lung.

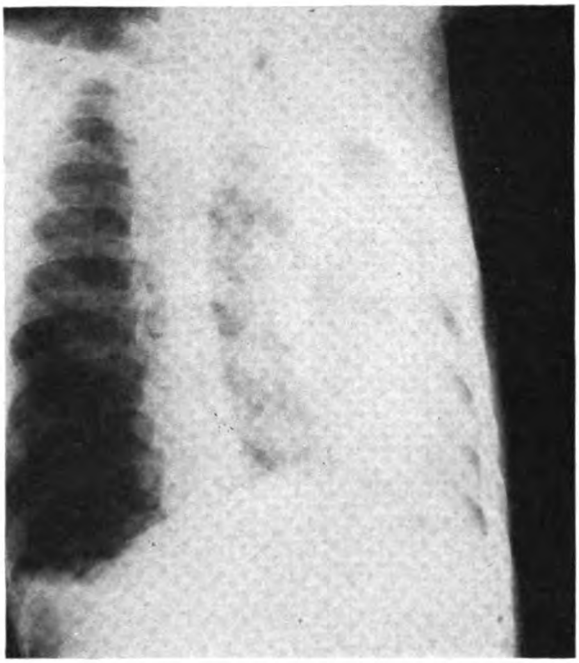

Fig. 56.—Film of worker shown in Figs. 54 and 55. Lateral view taken Dec. 14, 1943.

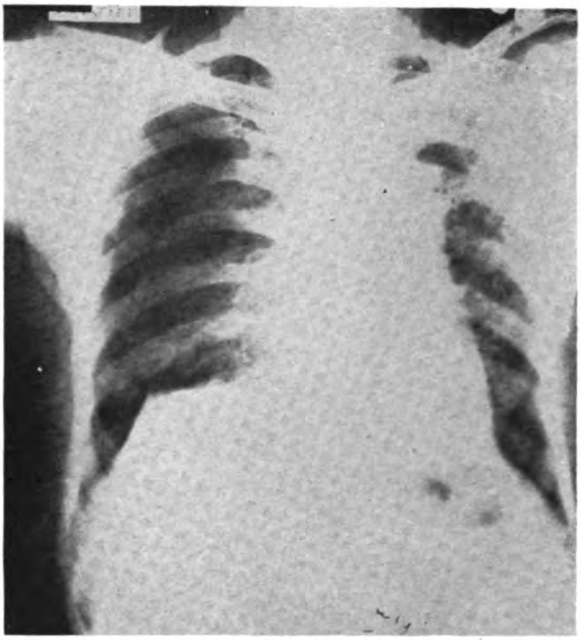

Fig. 57.—Film of worker shown in Figs. 54 to 56 taken Jan. 17, 1944. Expansion of right lung has occurred with only small area of collapse in upper right portion.

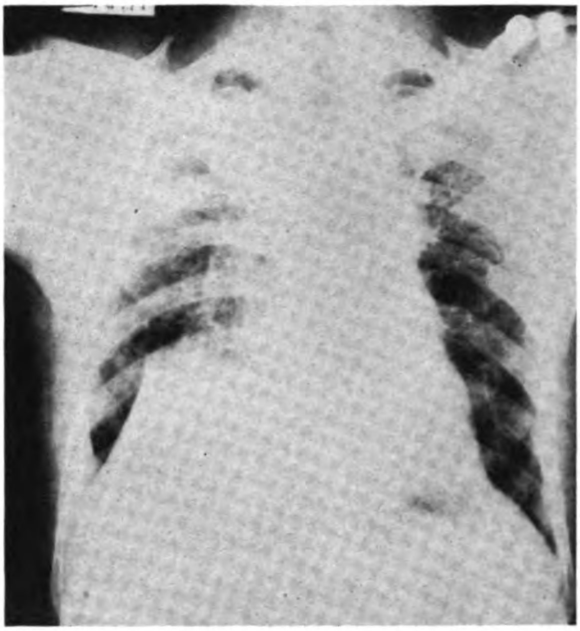

Fig. 58.—Film of worker shown in Figs. 54 to 57 taken April 3, 1944. Normal lung expansion with scattered area of fibrosis in both lungs.

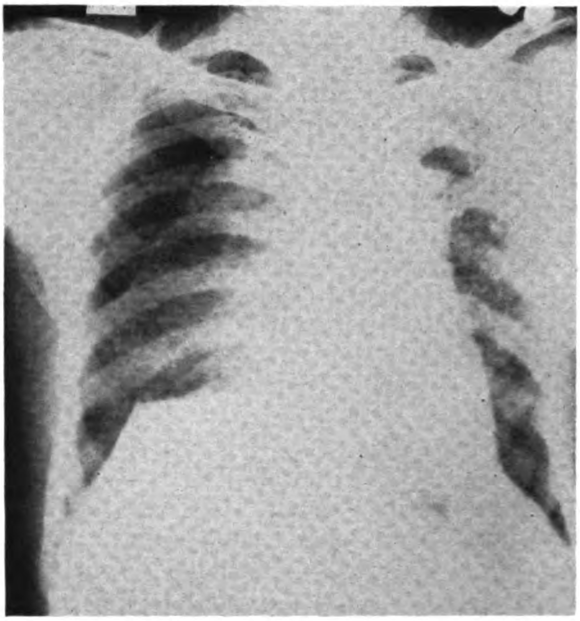

Fig. 59.—Film of worker shown in Figs. 54 to 58 taken May 2, 1944. Lung has again collapsed in right side with large emphysematous bleb present.

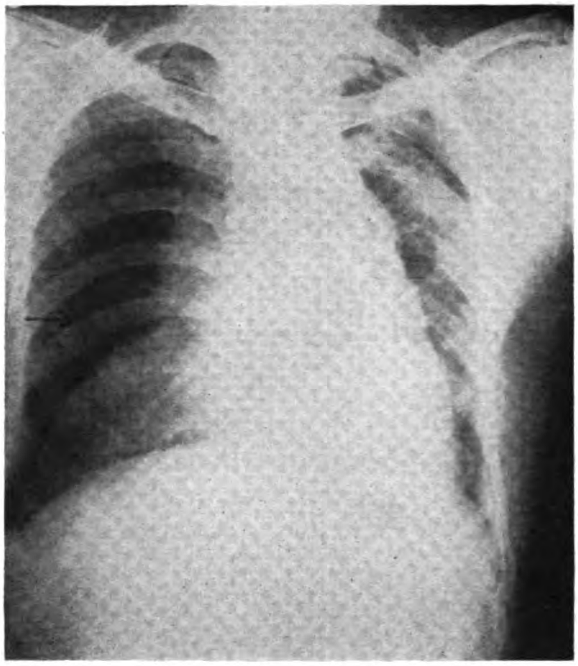

Fig. 60.—Film of worker shown in Figs. 54 to 59 taken June 11, 1944. Collapse of lung still present with large emphysematous bleb more distinct. Smaller blebs present on left lung with partial collapse of apex of left lung.

The patient remained at home and at rest until April 3, 1944, at which time an examination showed the lungs to have re-expanded. In May of 1944 he suffered another attack of breathlessness and the film of that date shows that the lung had again collapsed. It also demonstrates a large emphysematous bleb on the lung surface. The film taken in June shows this bleb even more clearly as well as some smaller blebs in the opposite side.

The physical examination of this man in December, 1946, is interesting. On that date his weight was 122 pounds; height, 5 feet, 7 inches; respirations, 22; pulse rate, 76. He was slightly cyanotic and looked miserable. The clavicles were prominent and expansion very poor. The percussion note was hyper-resonant with underlying impairment on the right posteriorly and laterally toward the base. The breath sounds were almost inaudible except on the right anterior mid-chest region. Irregular fine crackling and explosive rales were present over the posterior portion of the chest posteriorly as well as in the left interscapular area and left base.

## Diatomaceous Earth

In the introduction to the section of this book dealing with the dusts, it was mentioned that the diatoms, small aquatic plants, require silica for the formation of their skeletons or shells. Geologic studies indicate that for millions of years these silicious skeletons had been accumulating in the lakes and sea waters, existing during the Miocene Age, to form large deposits of material we now know as diatomaceous earth. Probably due to violent earth movements in prehistoric times, certain of these deposits became more abundant in some areas. On the west coast of California and Oregon there are regions rich in this material.

Of some 10,000 different species of diatoms, relatively few have any commercial value. The important ones are acicular or "needlelike" in form. In addition to the elongated form, another property of this substance is its exceedingly light weight. In finished form, ready for industrial application, it is fluffy. This lightness and fluffiness permits its wide use as filters, fillers, and absorbents; it is also used in paint, rubber, plastic moulding, packing and fire-proofing material, in insulators or as absorbents for acids, water, and impurities.

Crude diatomaceous silica is obtained by quarrying or open pit mining. Today large power shovels have largely replaced hand quarrying. After removing this earth, it is sent to the mill where, in a drying process, all moisture is removed. The material is handled with great care to separate the diatoms from each other without destroying their structure. Then the separated diatoms are conveyed by air stream through a system of cyclones, traps, and kilns and finally packed for shipment.

For many years the opinion has been prevalent that in the diatomaceous earth industry the workers are not subjected to the development of any disabling fibrosis. Recently, however, there seems to be some ground for questioning the innocuousness of this substance. Little information has ever been published regarding the effects of diatomaceous earth and for that reason the author intends to present at least an intimation of recent developments.

Some years ago, Dr. Leroy Gardner experimented with samples of diatomaceous earth. Following these experiments he found silicotic changes which he was at a loss to explain. At that time his analyses of the raw material had to be carried out chemically. Later, when he had the advantage of the x-ray diffraction method, he found that if diatomite is calcined with certain fluxes, a portion of the amorphous silica is converted into cristobalite, a crystalline silica which is the potent cause of silicosis. Upon making this discovery Gardner believed that the original experiments were made upon calcined diatomite which caused the silicotic reaction.

At Saranac, Gardner continued his experiments with the raw earth material to find that the diatomaceous earth is harmless like other amorphous silica. An inhalation exposure for a period of two and one-half years produced no fibrosis of the lungs. The reaction was confined to phagocytosis. He then injected the diatomaceous earth which had been calcinated with flux containing about 25 per cent cristobalite. It was noted that this caused a nodular fibrosis.

In 1945 Gardner came to California to study the diatomaceous earth industry, to review collective x-ray films of the workers, and to consult with physicians engaged in caring for these men. In viewing the films of men who had worked for years in this material, Gardner wrote later to one of us that "silicosis is conspicuous by its absence." He noted an appreciable amount of benign, nonspecific pneumoconiosis "as one would expect in any group exposed to dust of any kind." The x-ray pattern is that of exaggerated linear markings, and clinically the men with these changes exhibited no symptoms or disability.

In the collection of films reviewed by Gardner, there was a very small group in which the upper portion of the lung fields showed localized shadows which he interpreted as evidence of a chronic infection, presumably tuberculous in origin.

At the time of Gardner's death the status of the diatomaceous earth problem was simply this—an exposure to this substance did not produce true silicosis. To those of us on the Pacific coast it was becoming more and more apparent that we were dealing with a new entity. To help clarify the thinking on this point, a meeting was held at Santa Barbara in April, 1947. Among others, this meeting was participated in by Lanza, an authority in the field of pneumoconiosis and one who had conferred frequently with Gardner on the diatomaceous earth problem, by Karns of Stanford, an able pathologist who had also spent some years at Saranac, by Walter Anderson of Lompoc, who has cared for these workmen for many years, and by Reginald Smart, of Los Angeles, whose knowledge of the diatomaceous earth problem is exceeded by no one. From this meeting came the following observations:

1. The workmen exposed to the straight diatomaceous earth (not calcined) may reveal by x-ray evidence of an increase in the perivascular markings with degrees of fibrosis. This picture is similar to that seen in any workman who has spent some years exposed to a dust. In other words, the x-ray picture may be that of a benign pneumoconiosis and the workmen reveal no signs, symptoms, or disability.

2. Those who have been exposed to the calcined earth present an entirely different picture and one that is far different from that seen in silicosis. The x-rays are conspicuous by the absence of discreet nodulation. There is a marked increase of the perivascular markings and fibrosis. The x-rays give one the impression of an infection being present as suggested by the fuzziness and haziness. Pneumothorax is common.

The pathologic picture is that of marked fibrosis, shrinkage of the lung tissue, and contraction of the veins (as in contrast to the contraction of the arteries seen in silicosis). It was noted that the inhalation of the diatomaceous earth and its presence in the lung seemed to inhibit infectious processes, especially tuberculosis. Dr. Caldwell, of the Santa Barbara Tuberculosis Sanitarium, testified that in his opinion, if tuberculosis became superimposed upon this pulmonary affection, it was cured in shorter time than in the average individual. This, the reader will recognize, is in sharp contrast to the susceptibility of the individual with silicosis to tuberculosis.

The peculiar reactions within the lung of the diatomaceous earth worker appears to be due to the action of cristobalite, a crystalline silica, and, to a less extent, to tridymite. It is believed that these two substances become released or activated when subjected to high temperatures.

Much work remains to be done on the study of the affect of diatomaceous earth upon the exposed workman. This will take the combined investigation of the clinician, pathologist, roentgenologist, and possibly the chemist in the field of molecular structures.

### Tripoli

The effect of exposure to tripoli dust was considered by McCord[5] and reported by him in 1936. In 1943 McCord, Meek, and Harrold[6] added considerable information to McCord's original study. It appears that the term "tripoli" has been used to designate a form of free silica which is soft, porous, and friable. Tripoli is not to be confused with diatomaceous earth. The x-ray diffraction pattern of tripoli is that of quartz. Being a quartz containing approximately 98 per cent free silica, it would be expected to produce a high incidence of silicosis among those who work with it. But on the contrary, the incidence of silicosis from exposure to tripoli is relatively infrequent.

McCord and co-workers concluded that there are in existence certain deterrents which inhibit the injurious action of tripoli, namely:

"1. The high canaliculation (porosity) of true tripolis eventuates in a large surface area per unit of mass (specific surface).

"2. High specific surface favors adsorption phenomena of which hygroscopic films are one aspect.

"3. Related to surface phenomena is an unusual degree of dipolar electrical attraction. A dipole is a positive and negative "free charge" fixed in one mass unit, with a total charge of zero, but having attraction for other dipoles on other units or on neighboring surfaces. Tripoli does possess extensive electrostatic charges "true charges," but this is equally true of quartz silica and other dusty materials with behavior different from tripoli.

"All the foregoing natural properties operate to provide a measure of coherence between fine tripoli particles so that a relatively greater degree of resistance is offered to those forces that tend to thrust fine materials into the atmosphere as suspended dust. Once in the atmosphere, dipolar attraction is markedly diminished as interparticle distance increases.

"4. The fourth protective feature is artificial. Adsorption forces leading to hygroscopic films on particle faces necessitate the waterproofing of tripoli prior to use as a foundry parting. The chemical nature of the waterproofing coating favors no, or diminished, physiologic reaction. These materials have melting points well above body temperature and are not known to be removed by any physiologic process. The extent of physiologic inertness appears to depend upon the efficacy of the coating process. Not all, and particularly not all particles below 5 microns, are coated.

"Recognizing tripoli as a highly silicious substance and thus potentially dangerous when in a suitably fine state, these four-mentioned factors are marked as contributors to the low incidence of silicosis among those workers engaged in its manipulation."

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# CHAPTER XXXII

# THE INERT DUSTS

In the portion of this book which is devoted to metals, it was noted that the dusts or oxides of certain metals, such as lead or manganese, may, when inhaled, exercise a profound systemic effect. Others, such as cadmium or beryllium, may cause a chemical pneumonitis. These toxic dusts are, however, relatively few in number. After excluding these toxic dusts, as well as silica and asbestos, there remains a large group of organic dusts which are inert or harmless. An explanation as to the reason of their harmlessness is in order. Three prime reasons for their innocuousness are as follows:

1. *The anatomic-physiologic mechanism* of the *respiratory tract.* That only negligible amounts of dust ever reach the lungs is a fact not realized by the layman and frequently forgotten by physicians. The efficient barrier which nature has devised enables man to go through life without incurring harm, although he may be constantly living in a dusty atmosphere. The first obstruction encountered by dust is the fine hair at the port of entry, the nose. These hairs filter out the larger particles of dust. Those smaller particles which pass this first barrier are then met by the mucus of the upper respiratory tract where they are engulfed to be blown or coughed out. In the trachea and bronchi the ciliated epithelium are in constant upward motion and propel fine dust particles to the point where they are coughed out or expectorated in the sputum. Beyond this barrier, the phagocytic cells gather in dust particles and transport them to the hilar nodes to be destroyed.

2. *Particle size.* It should be obvious that for any dust to be inhaled it must be floating in the atmosphere. To be suspended it must be almost invisible, extremely small, and light weight. Thus any atmosphere in which the particle size of the dust is so large that it is plainly visible is not likely to be dangerous, whereas, on the contrary, a room appearing to be only slightly dusty may be dangerous.

Not infrequently, workmen will claim that such a trade as grinding metal has affected their lungs. If the examining physician will ask them to bring in some of the dust and will then toss this dust out in front of the workman to demonstrate how these particles immediately fall to the floor, the demonstration invariably convinces the workman of his erroneous assumption.

3. *Organic dusts are chemically inert.* If a portion of any one of these inert dusts reaches the pulmonary tissue, no chemical reaction occurs. This fact was mentioned in the opening paragraph and needs no further elaboration.

It is not contended that after many years of exposure certain dusts will fail to produce what is known as a benign pneumoconiosis. As aptly defined by Pendergrass and Leopold,[1] benign pneumoconiosis is a term used to designate that condition in the lungs which results from inhalation of organic or inorganic dusts which, while they may be retained, are neither toxic, allergenic, nor pathogenic. Although they may be capable of producing an insignificant amount of reactive fibrosis, this condition never progresses to true nodulation. Unlike free silica and asbestos, the presence of these dusts produces no symptoms, no disability, and *no predisposition to tuberculosis.*

The foregoing truth cannot be emphasized strongly enough. The presence of some degree of fibrosis is indicated by the x-ray films, but it should not be interpreted as a disease. It was Pancoast[2] who said: "Let's not talk about normal chests. We see normal chests probably only in children. As individuals grow older you will find all sorts of shadows in their chest x-rays. Let's speak of the healthy chest." This admonition should be remembered by industrial physicians as they read the films of men exposed to the inert dusts, or even to silica. The inexperienced physician should not commit himself but rather seek the opinion of an expert in the interpretation of films of those exposed to dusty trades.

**Siderosis.**—After a number of years of exposure, the inhalation of iron-containing dust will cause a pigmentation on the lungs. This is a benign condition. X-rays will reveal round shadows throughout both lung fields. The rounded shadows have often been falsely interpreted as silicotic nodules. Sander[3] states that: "The x-ray appearance of the iron deposits differs from silicosis in that the hilum shadows are not enlarged, the pigmented shadows are more discrete and sharply defined than silicotic nodulation, and there is no tendency to confluent nodulation." In cases where the x-ray films cause some doubt as to the etiologic cause, the history of occupation should be the deciding factor.

In 1944 the author, while in Cleveland, had the privilege of reviewing the x-ray films of a large group of welders who had been working on jeeps for the army. These men were all healthy and had no complaints regarding any hazardous work. However, the rejection of several of these welders by the army led to the x-raying[4] of eighty-three welders in one plant, fifty-nine of whom showed varying degrees of siderosis, which, in a few, was quite marked.

It is believed that it takes upward to eight years to bring about a recognizable degree of siderosis and then usually only after the concentration has been fairly severe. But because these markings are pigmented and because the deposits do not produce fibrosis, there is no functional impairment. Likewise, siderosis does not predispose to tuberculosis or to reactive latent tuberculosis.

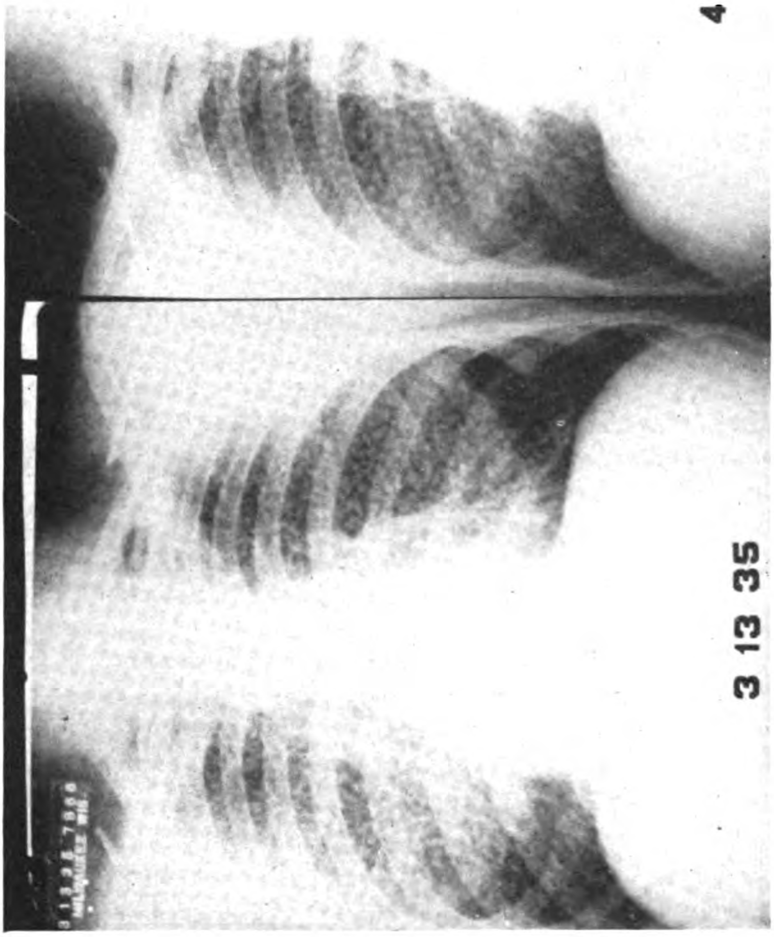

Fig. 61.

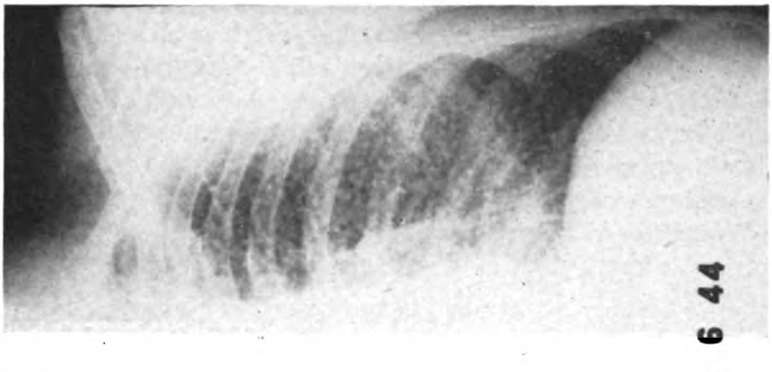

Fig. 62.

Fig. 61.—Film of welder showing pigmentation due to iron dust.
Fig. 62.—Film of worker shown in Fig. 61 taken nine years later. No change noted in x-ray film. Patient symptom free.

Figs. 61 and 62 were taken nine years apart. For a period of eleven years prior to 1933, this workman had spent 25 per cent of his time inside tanks, as a welder, using uncoated electrodes. Since 1933 he has done no welding. He has been entirely free of respiratory diseases or symptoms, is a long distance swimmer, and at the age of 45 years is able to swim as far as he did ten years previously. His occupational history reveals no exposure to any type of dust or fume other than iron oxide. Periodic chest films have shown no change in the lung shadows in the past nine years, and he has had no symptoms referable to the lungs.

**Byssinosis.**—This term has been applied to a peculiar respiratory affection resulting from exposure to cotton dust. In 1941 the author reported[5] a case of asthma with unusual x-ray findings in a cotton worker. Prior to that time the American medical literature was devoid of reports of specific cases of a similar nature. Much of our knowledge regarding the effects of cotton dust has come from English investigators.

The reader may think it odd that this dust is considered under the heading of the inert dusts, but it is an inert dust within the meaning of that term; that is, it causes no appreciable fibrosis which in turn could cause disability. The illness is due to allergens, or fungi—not to pulmonary fibrosis.

It is the consensus of opinion that cotton dust causes no appreciable fibrosis. However, some fibrosis was present in the author's case. Bolen[6] also noted fibrosis in several of his cases. Ritter and Nussbaum,[7] however, after an intensive survey of cotton workers in Mississippi, failed to find any x-ray changes in the lungs of these workers. In their initial investigation Ritter and Nussbaum[8] encountered a bacteria identified as aerobacter cloacae which substantiated the earlier findings of Neal and associates.[9]

SIGNS AND SYMPTOMS.—*Respiratory Effects.*—According to the English investigators, the effects produced by breathing an atmosphere heavily impregnated with cotton dust are characteristic and indicate a severe irritation of the air passages. Dyspnea is a prominent symptom, but it is dyspnea of an undoubtedly asthmatic type. It is different from the dyspnea of silicosis, which involves the abdominal efforts and not the marked thoracic efforts of those affected by cotton dust. The cough is dry with little expectoration. Whatever sputum is produced is of an extremely tenacious character. The attacks tend to disappear over the week end or during a vacation, but reappear with the resumption of work. Chronic asthma eventually results in loss of weight, increased dyspnea, emphysema, and the development of the barrel type of chest. From reading the English literature it can be assumed that it takes from twenty to twenty-five years for the development of severe symptoms. It is also noted that the coarser grades of cotton contain more dust than do the finer varieties and, therefore, constitute a greater hazard. In addition to the asthma and bronchitis, catarrh of the upper respiratory passages is common.

Almost all writers on the subject give a fairly similar description of the symptoms involved. After the worker has remained for many years free from perceptible symptoms apart from a slight cough, there suddenly occurs increased coughing and attacks of dyspnea. In general, these attacks occur on Mondays, and during the rest of the week the worker remains in good health. This "Monday fever" slowly increases in frequency, spreading gradually over the following days of the week, until finally the worker suffers from permanent trouble. Cessation of work brings about improvement, but resumption of work is followed by increased ill-health which is often of rapid evolution, though sometimes progressive, so that finally the worker is obliged to give up his work altogether. This appearance and disappearance of the disease is definitely characteristic of the allergic nature of the affection.

*Allergic Phenomena.*—These have been reported by the English investigators as well as by Teleky in Vienna and Schmidt in Germany. Weavers do not appear to be affected by this allergic reaction as much as those who prepare the material. In the latter group, irritation of the upper respiratory passages, a sensation of oppression of the chest, and attacks of coughing with scanty sputum are the common complaints.

*Fibrosis.*—Autopsies in cotton strippers and grinders fail to reveal pulmonary fibrosis. It is thought by most investigators that any fibrosis developed by such cotton workers is the result of a combination of bronchitis and asthma and not of the cotton dust itself. In England, the Home Office Departmental Committee defined byssinosis as a chronic bronchitis complicated by emphysema in its later stages and by asthma in some cases. It is felt by these investigators that the combination of asthma and chronic bronchitis is due to the action of the protein fraction of the cotton dust inhaled.

*Nonpulmonary Condition.*—Certain nonpulmonary conditions may exist in the industry. Workers in the processing rooms may develop conjunctivitis or blepharitis (from the burned products of the gassing of double yarn), headaches, lassitude, and loss of appetite (from the high temperatures), in addition to the possible effects of carbon monoxide.

*Molds and Fungi as Factors.*—A comment by Collis[10] particularly interested the author in view of the x-ray findings in the case he is about to report. After investigating the cotton mills of England, Collis concluded that the occasional severe epidemics of coughing among these workers were due to mildew and molds and that these molds were *Penicillium glaucum* or *Aspergillus glaucus.* Others have indicated a relationship between the chest conditions and fungus. The British statistics show a high incidence of upper respiratory diseases in persons engaged in the preliminary operations such as balers, carders, or sorters. Our patient was a sorter for twenty-eight years.

CASE 1.—A 40-year-old Negro man presented himself to the writer with a complaint of attacks of nocturnal dyspnea.

*History.*—Since the age of 12 years this patient had worked as a cotton sampler. He had not worked in the cotton mills or been connected with any other processing phase of the cotton industry; his activities had been confined to the handling of cotton for inspection. The only time he was not exposed to this was during a brief sojourn in the army during World War I. Until 1918 his work was done in the South, and since that time he had been engaged as a cotton sampler in Los Angeles.

About one year previous to this examination he noticed a sensation which he described as a dull weight or oppression between the shoulder blades. He claimed that this was not a pain and never had felt like a pain but was simply a heavy sensation in the upper posterior chest.

Six months after noticing this heavy feeling he began having nocturnal attacks of shortness of breath. He described these attacks as follows:

> "I do not have any trouble during the day or while I am at work. I am all right until after I have been asleep for a while. I usually fall asleep without difficulty, but about an hour later, or sometimes less, I am awakened by a wheezing sensation in my chest. I then start coughing and get very short of breath. I feel as if my air is being cut off, and I have to get up. When I get up I usually spit a heavy, thick, sticky substance, and then I start to feel better. I never notice any of this whistling or wheezing in my chest during the day and I rarely cough during the day. I do not have any shortness of breath except if I walk about three blocks, or if I climb a flight of stairs in a hurry."

*Symptoms Referable to the Various Systems.*—Essentially negative.

*Past Medical History.*—Entirely negative with the exception of gonorrhea contracted while in the army, at which time he also developed a sore at the end of the penis, to which he gave the familiar term of "hair cut."

*Physical Examination.*—The patient was a Negro man; height, 5 feet 8 inches; weight, 191 pounds. He was 40 years of age. His intelligence was above the normal for his race.

The pupils reacted to light and accommodation; the eyegrounds were normal. The throat was normal; the tonsils, atrophied. The teeth appeared to be in a good state of hygiene. The neck was short, bullneck type.

The chest was barrel-shaped, measuring 41 inches. Following full inspiration it measured 42 inches. There was a moderate limitation of the normal excursion rate bilaterally. There was no noticeable lag on one side over that of the other. Percussion note was slightly duller than normal. Auscultation of the lungs revealed the breath sounds over the entire chest to be quite distant, almost imperceptible. In spite of the history of wheezing and whistling sounds, we were unable to detect any râles during three separate examinations of this patient's chest. The heart rate was 84, the sounds were distant, no murmurs were heard. Blood pressure: right arm, 128/90; left arm, 130/90.

The abdomen was obese; no organs or masses were palpated. Genitals and rectal examination were negative.

*Neurological Examination.*—The pupils reacted as noted; knee jerks, 2 plus; Achilles reflexes, positive bilaterally; Romberg sign, negative; finger-to-nose test, negative; Babinski test, negative.

*Laboratory Examination.*—Urinalysis was negative; blood count, normal; blood Wassermann, 4 plus; Kahn, 4 plus; repeated sputum specimen, moderate number of white blood cells; ordinary bacterial flora; acid-fast bacilli not found; no fungi found; guinea pig, negative for tuberculosis.

This case presented the following similarities to the English cases:

1. Length of exposure. Twenty to twenty-five years is the time interval the English have observed before the pronouncement of severe symptoms. Our patient had an exposure of twenty-eight years.

2. The symptoms. Cough with scanty but tenacious sputum (the laboratory technician had to cut specimens in this instance with scissors for material for slides); oppression in the chest (this was one of the first things noted by our patient); dyspnea at night with the presence of râles; symptoms more marked in those who handle the raw cotton (our patient was a cotton sorter).

3. The development of emphysema and barrel type of chest.

4. The possible infestation of fungus. This question had to be considered in our case.

*Roentgenographic Interpretation.*—The chief problem was to evaluate the chest plates. These were so strikingly unusual and outside the experience of several clinicians and roentgenologists who viewed them that they were mailed along with the history to several nationally known authorities for their comment. In the base of both lung fields, especially on the left, there were concentric whirlpools as if made by interlacing white threads. These films in stereo were interpreted by our roentgenologist as follows:

X-ray examination—stereo of the chest (Fig. 63): There is a definite area of increased radiability along the upper and lateral wall of the left lung field, consistent with a partial pneumothorax or coalition of several large emphysematous blebs. On the right side there is also noted near the apex and along the upper lateral wall an area of increased radiability suggestive of a similar condition. The low, flat diaphragm, together with a rather enlarged chest, bears out the picture of general emphysema. There is increased fibrosis around both lung fields near the base of the heart on the left side, but the cause of this is rather obscure. It is very definitely not suggestive of pulmonary tuberculosis, nor does it have the markings of pneumoconiosis. This fibrosis is most likely due to a chronic bronchitis with a coexistent pneumonitis. It is conceivable that these rounded areas of increased density might represent small areas of atelectasis developed on a basis of emphysema plus a partial pneumonthorax.

The nearest approach to similarity of x-ray findings to the author's case is that of McCarthy and Akenhead[11] in which they encountered patches of increased density in both lung fields.

Dr. Leroy Gardner, in a personal communication, at Saranac, commented:

"I am most grateful to you for letting us see the films of your Negro cotton sampler. Both Dr. Sampson and I have studied them with greatest interest. I have been looking for a long time for such a case, as I have never had opportunity to study any material after long exposure to dust of this kind.

"We quite agree with you that the anatomic charges are probably not due directly to exposure to cotton dust, that there is a complicating infection, but whether this is directly attributable to the dust exposure or not cannot be established.

"Dr. Sampson saw the films before he read your letter, and because they were marked California he naturally thought of coccidioidal granuloma as a possible etiologic agent of infection. This I would assume you have excluded.

"I certainly feel that the sputum should be examined for all kinds of fungus and unusual bacteria. I quite agree with you that lues can probably be excluded as a cause of pulmonary change."

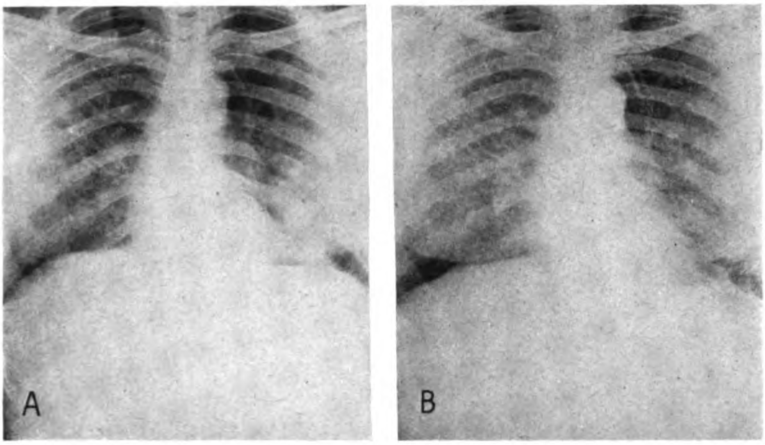

Fig. 63.—These films are of the Negro cotton worker referred to under Byssinosis. For their interpretation, the reader is referred to the comments in the text. The film shown in *B*, taken almost one year after that presented in *A*, shows no perceptible change.

Dr. H. L. Sampson, at Saranac, stated:

"Definite pathologic process involving both lungs, more particularly at the base, and more particularly the left base, with some emphysema in the top of each lung. The character of the shadows suggests this to be nontuberculous, but it does not have the nodulation characteristic of silicosis. About the best that can be said for the process is that it does not appear to be tuberculous."

Dr. Clarence O. Sappington, after reviewing these films, concluded that: "It is my opinion that the greatest possibilities in this case lie between a so-called mixed infection and some sort of fungus infestation."

From the various opinions expressed regarding the x-ray films, it is evident that the markings indicate (1) a pronounced emphysema and increased fibrosis; (2) the presence of snowball-like or whirlpool configurations of unusual and undetermined character; (3) the possibility of fungus infection; (4) the possibility of gumma.

The maintenance of weight, the absence of afternoon temperature, the negative sputa, the negative guinea pig inoculation, and the character of the films argue against the possibility of *tuberculosis*. *Syphilis of the lung* can definitely be ruled out. At the time this patient was first seen the positive Wassermann was noted, and the patient immediately underwent antisyphilitic treatment. Examination nearly one year later showed the treatment had changed the complexion of the Wassermann reaction, but the chest films remain unchanged and, if anything, seemed further accentuated. According to Sante,* the lungs are structures relatively, if not entirely, immune from syphilis. Our present knowledge of the action of inert dusts dispels any likelihood that the markings of the lung as revealed by x-ray are the result of cotton dust. It is conceivable that the repeated attacks of allergic asthma, in conjunction with continuous inhalation of dust over many years, may finally set up pulmonary changes consistent with a chronic bronchitis and produce a loss of elasticity of the pulmonary tissue.

This patient is no longer exposed to cotton dust. He has been followed and examined periodically. He is symptom free but his chest films remain unchanged.

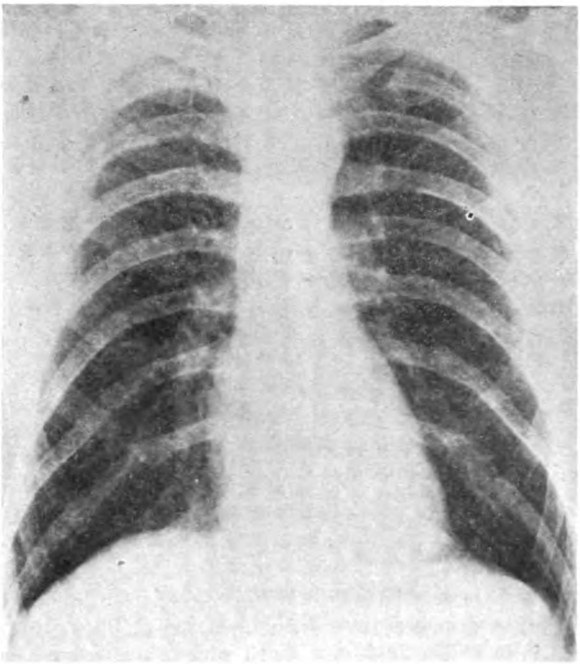

Fig. 64.—Film of a 35-year-old male who worked in cement dust for twenty years. (A. J. R. Curtis, of Portland Cement Co., and Dr. Gardner and Dr. Sampson, of Trudeau Sanatorium.)

**Cement Dust.—**

The essential ingredients of Portland cement are $CaO$, $SiO_2$, $Al_2O_3$, and $Fe_2O_3$. The chief raw materials which supply these constituents are limestone to furnish the $CaO$ and shale to supply the other ingredients. An analysis of Portland cement reveals that it contains 19.86 per cent of $SiO_2$, yet almost none of this silica exists in a free state. Complete studies were made in eleven plants involving 2,278 employees. Of these, 299 worked in a dust-free atmosphere.

CLINICAL FINDINGS.—Complete physical examination of 2,278 employees in eleven different plants situated in widely scattered parts of the country has demonstrated nothing even suggestive of a pneumoconiosis. Stereoroentgenograms of the same group have revealed certain variations of the usual pulmonary shadows. The same abnormalities also occur in the films of other industrial groups and are in no sense specific for these (Figs. 64 and 65).

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* Sante, L. R.: The Chest Roentgenologically Considered. Annals of Roentgenology, vol. XI, New York, 1930, Paul B. Hoeber, Inc., p. 386.

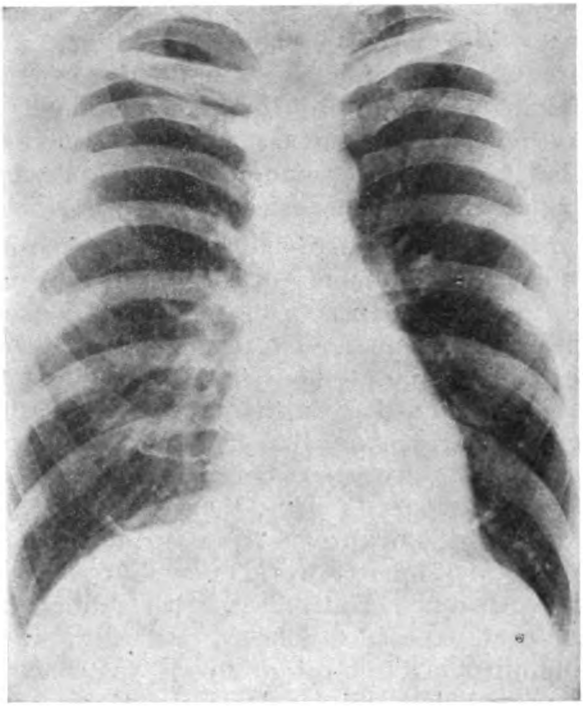

Fig. 65.—Film of a 55-year-old male who worked for over thirty years in cement dust. The linear markings are comparable to one of this age not engaged in a dusty trade. This and Fig. 64 serve to illustrate the relatively minor extent of fibrosis resulting from exposure to the inert dusts. (A. J. R. Curtis, of Portland Cement Co., and Dr. Gardner and Dr. Sampson, of Trudeau Sanatorium.)

The Saranac group quite thoroughly investigated the relationship between dust exposure and tuberculosis, bronchitis, and pneumonia, and their study indicated that the incidence of tuberculosis and other pulmonary infections is as low or lower than that in the general population. There is nothing to suggest that prolonged inhalation of cement dust has altered natural susceptibility to tuberculosis. They likewise found no significant effect of this inert dust on old tuberculosis. It was also noted that the incidence of bronchitis and pneumonia in those working in a cement dust varied, as did the figures for the general community where the various plants were located. The reports of a low incidence of bronchitis in the dry sections of Texas or Kansas, as contrasted with the high incidence in one of the dampest parts of the State of New York, suggested a marked clinical influence. Absenteeism due to upper respiratory infections such as coryza was somewhat greater than that in the general community.

**Calcium and Magnesium Carbonates.**—From these compounds come limestone rock, marble, chalk, and calcined magnesium used for insulations, furnace linings, and the like. Men have been exposed to these dusts for years, but there is no evidence that they are harmful to the lungs.

**Carbon Dust.**—This dust is a common constituent of the air in any industrial district where anthracite or bituminous coal is used. Exposure causes black pigmentation of the lungs, noted in city dwellers even though they have no specific or intense exposure. Miners reveal a fibrosis greater than that of the nonexposed, but it is a nondisabling fibrosis, unless the carbonaceous substance is combined with silicious material.

**Tobacco Dust.**—Tobacco dust has been referred to in the literature of the past as a cause of pulmonary disease. In reviewing this literature it is interesting to note that practically all, if not all, of these reports were made without the benefit of x-ray. Some of them were purely assumption, such as the observation that tuberculosis was greater in tobacco workers of Baden than in the general population, and it was, therefore, concluded by Stephani that the tobacco dust was responsible. The probability of faulty hygiene, poor working conditions, long hours of labor, etc., was overlooked. Thus there crept into the literature a condemnation of a dust not proved to be an actual offender. Long,[12] after studying a group of tobacco workers over a period of ten years, did not once see the occurrence of this disease. He studied 2,254 persons, of whom 1,246 had been exposed to tobacco dust while working. By comparison of the findings of these two groups it was concluded that pneumoconiosis (tabacosis) does not develop in tobacco workers. The value in this observation lies in the x-ray and fluoroscopic revelations as compared with the absence of these in other reports.

**Mill Dust.**—While these dusts have no direct effect upon the tissues of the respiratory tract, they do carry certain allergens. In various mills, factories, and plants, the concentration of inert dusts may be very great and yet not affect the majority of workmen employed there. A few of these, however, may develop a respiratory or cutaneous allergy, the cause of which may be unrecognized by themselves or their physicians. Particularly is this likely to be true in those who work in or around flour or grain mills. Attention to this problem has been called by the report of Wittich and Stackman.[13]

**Discharge of Dust Into Air.**—Wittich and Stackman[13] point out that in the milling of grains the problem becomes one which concerns not only the workmen in these mills, but also the community, for the dust discharged into the air from "cyclones" atop these mills causes high contamination of the air over an area of many blocks. In fact, the dust hazard for the workmen is often controlled while the general community remains unprotected. This investigator has pointed out that slides exposed for twenty-four hours inside offices within six blocks of these mills showed numerous grain smuts, rusts, and molds. It appears that the worst offenders are the grass and grain smuts.

SMUTS.—These are minute, threadlike, parasitic plants of mycelium, which enter the growing portion of the seedling and grow up with the host, remaining invisible until the heads of grain appear, when they partially or wholly destroy the heads, appearing at that stage as masses of dark substance. These masses contain spores which further propagate by getting on or into the seeds, where they re-enact their life after the seed is planted. Due to these smut spores, contact dermatitis, atopic forms of skin allergy, perennial rhinitis, and asthma may occur. These conditions may be fairly abundant wherever conditions favor the infestation of smut. They are most pronounced in the Middle West, the Pacific Northwest, the South, and the Southwest.

**Grain or Malt Fever.**—There exists another illness among grain workers which, when described by them, sounds fantastic. This condition, referred to as "grain fever" or "malt fever," came to the writer's attention just after the printing of this book had begun, so that there was not sufficient time for thorough laboratory investigation of this type of case.

A weighman at the grain elevator of a malt company in Los Angles was hospitalized suffering from a penetrating wound of the chest. In relating his past medical history he described recurrent attacks of an odd malaise which he attributed to exposure to malt dust. The other men at the mill were subsequently reached, and the patient's history was substantiated.

SYMPTOMS.—A description of this illness follows: Several hours after leaving work, or while asleep, the workman becomes aware of a headache, weakness, fever, chills, and cold sweats, as well as nausea, cough, and often vomiting. By morning these symptoms have all subsided, the victim feels normal, and reports for work. Such nocturnal attacks may continue for several weeks. This strange illness occurs most frequently among men never before exposed to grain dust, or among experienced men at the beginning of a new season or upon returning to work after a short absence. In this respect it is not unlike metal fume fever, an illness to which only those constantly exposed appear to develop an immunity. No satisfactory explanation can be offered for this reaction to malt dust, except that it is probably due to a foreign protein.

Symptoms identical to those found in our case were reported in a study of grain handlers at Buffalo.[14] The exposure caused no disability or chronic illness.

*Bagassosis.*—Bagassosis is a term applied to a syndrome resulting from exposure to bagasse, the residuum of sugar cane after the sugar has been extracted. The substance is used in the manufacture of insulating boards. In the process, bales of bagasse are crushed by machinery with the evolution of much fine, dry, dust.

After several months of exposure to this dust, men develop rather suddenly a cough, productive of a foul-smelling sputum, dyspnea, and often retrosternal pain. This condition continues for several months after removal from exposure, but eventual recovery takes place. It is believed that the cause of this pulmonary disturbance is due to an antigen in the bagasse. No definite roentgen picture has as yet been established.

**Mica Dust.**—The chemical composition of mica has been mentioned earlier in this part of the book. It has been recognized that mica miners show x-ray markings suggestive of silicosis, but this is probably due to the presence of free silica in the earth from which the mica is procured. Whether or not mica *per se* causes a chest pathology similar to silicosis has not as yet been established.[15]

*Fibrous Glass (Wool Glass, Fiberglas).*—The name and the physical characteristics of Fiberglas are such that workmen are led to believe that this substance could be harmful to the lungs. Chemically, Fiberglas is similar to mineral wool and contains no free silica.

In order to establish the fact that Fiberglas is harmless to the lungs and causes little or no skin reaction among experienced workers, the manufacturers requested a thorough investigation as to the accuracy of the allegation that Fiberglas produces bodily injury.

Study of this occasional irritating effect upon the skin comprised a phase of the investigation undertaken by Dr. Siebert on behalf of St. Louis Local No. 1 of the Heat and Frost Insulators and Asbestos Workers.

An examination of the wrists, arms, ankles, necks, eyes, shoulders, and waists of approximately two hundred employees in the Newark Fiberglas factories were made by Dr. Siebert. These workers were employed on production lines and on fabricating operations, cutting, sawing, shaping, transporting, and packaging glass thermal insulating wool, both bonded and unbonded.

No case of eye irritation or conjunctivitis was discovered during this inspection. Evidence of significant skin irritation was shown by only one worker, a man who had begun work in the plant four days before. Two biopsy specimens of skin were removed from the arm of this newly employed worker.

Serial microscopic sections of this tissue examined by Dr. Siebert showed a mild degree of capillary dilatation in the corium of the skin. In addition, there were a few scattered lymphocytes around several of the dilated capillaries. The epidermis was entirely normal. No glass particles were found. Dr. Siebert reported no evidence of fibrosis, bacterial infection, allergy, or hyperkeratosis.

Several of the serial sections revealed a small amount of a brownish pigment in the corium which showed no inflammatory reaction around it.

> "It is assumed", says Dr. Siebert, "that this pigment is bakelite, one of the substances used as a binder for the glass fibers. The fact that there was no foreign body reaction around this indicates that this material is an inert substance and that it does not produce either a foreign body reaction or a fibrosis. The extremely mild inflammatory reaction in the corium of the skin would result from any slight mechanical irritation."

Dr. Siebert summarizes his conclusions regarding this phase of his investigation in the following words:

> "The author [Dr. Siebert] believes the following generalization is warranted by the evidence: contact with glass wool may cause ephemeral discomfort among workers inexperienced in handling the material. The skin irritation, however, cannot be regarded as a progressive dermatitis, for it is nontoxic and usually disappears in a few days. In extremely rare instances, where a worker may be predisposed to secondary effects of mechanical skin irritation because of idiosyncrasy, allergy or the presence of foci of infection elsewhere in his body and otherwise dissociated from the effects of mechanical irritation, lesions may persist. But these cases are encountered with such rarity that they do not justify incriminating glass fiber as a causative factor."

During the spring and summer of 1942, Dr. Marion B. Sulzberger, Assistant Clinical Professor of Dermatology and Syphilology, New York Post-Graduate Medical School, and Dr. Rudolf L. Baer, Junior Assistant Dermatologist and Syphilologist, New York Post-Graduate Hospital, carried out an experimental investigation of the effects of Fiberglas on the skin of laboratory animals and human volunteers. A report of their investigation was published in 1942.[17]

The laboratory animals used were rabbits and guinea pigs. Depilated sites on the animals were rubbed almost daily, except Sundays and several Saturdays, over a period of a month, with Fiberglas materials in both their wool-like and textile forms. Ten human subjects were rubbed daily, except Sundays, with the same materials for periods ranging from seven to nineteen days.

Histologic examination of the reaction sites in animals and human subjects showed evidence of a superficial inflammatory process. The histologic sections did not show any evidence of permanent damage, and the epidermis was generally found to be intact and undisturbed in its continuity.

Special methods of fixation which were designed to retain clinically visible glass spicules sticking into the skin were used in the attempt to demonstrate these spicules in the histologic section. However, in none of the many slides examined could the glass spicules be seen.

"This", Dr. Sulzberger and Dr. Baer say, "together with the fact that no disturbance in the continuity of the horny layer of the epidermis was seen, suggests that the spicules which were seen clinically after the rubbing were only very superficially embedded. No foreign body reaction was seen in any of the sections examined, thus demonstrating that no fragments of the various materials used in these experiments had penetrated into the deeper layers of the skin."

Summarizing the results of the investigation, Dr. Sulzberger and Dr. Baer say:

"All of the reactions observed were of a transitory and superficial nature. The only part of the reactions which persisted for more than a few days were slight thickening and brownish pigmentation of the skin. The latter were sometimes apparent for several weeks. No reactions or symptoms outside the rubbed skin areas were observed at any time during the experiment.

"The pieces of material which were seen sticking into the skin after rubbing with fiberglas in its wool-like form appeared to be very superficially embedded. This observation was borne out by the fact that ordinary washing with water easily removed a large part of the spicules remaining in the skin after rubbing.

"No definite sensitization to any of the materials could be demonstrated. In a few subjects there were a few mild and evanescent reactions to several of the materials, but it appeared very questionable whether these reactions were based on specific sensitization."

Tests conducted by Dr. Leroy U. Gardner, Director of the Edward L. Trudeau Foundation for Research and Teaching in Tuberculosis, have demonstrated that Fiberglas has no injurious effect upon the lungs.

In the annual report of the Foundation's Saranac Laboratory, for the year 1940,[18] the following statement appears:

"The apparent lack of pulmonary damage from exposure to glass wool dust is being demonstrated by an inhalation experiment. For over a year animals have now been exposed to as high a concentration of this material as could be obtained, with no serious effect upon their lungs. The dust has been produced by breaking into short lengths the finest textile glass (fiberglas) that is made, but none of the fibers that are floating in the air can be found in the lungs of the exposed animals. Their tendency, unlike that of asbestos, to form felt-like masses upon any object with which they come in contact probably prevents their inhalation. Slight amounts of chronic inflammation similar to those observed with other harmless dusts have resulted from accumulated particulate debris. These very fine, nonfibrous particles are the by-product of the violent beating used to break up the material."

"Exposure to the dust of glass wool, a material commonly used to insulate buildings, involves no hazard for the lungs because this fibrous material is not inhalable."

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# CHAPTER XXXIII

## TUBERCULOSIS AND PNEUMONIA IN INDUSTRY

In the opening paragraphs of this book the objectives of industrial medicine and its aims for the future have been stated. But work on certain of these objectives has not as yet been inaugurated. Other goals have been attacked for several years, the most notable of which is the campaign to detect tuberculosis in the industrial population, and to offer medical care for those found to be suffering from active tuberculosis. The National Tuberculosis Association, the United States Public Health Service, and the Trudeau Foundation for Tuberculosis at Saranac, New York, have taken a most commendable leadership in this campaign.

The promotion of a fact-finding survey for the incidence of tuberculosis in industry has not been conducted without certain opposition. In some instances management has been reticent lest the results might induce lawsuits to obtain compensation. On the other hand, labor has sometimes opposed these surveys, contending that it violates the rights of labor. Some private physicians have objected to the program, maintaining that it constitutes an infringement of the private practice of medicine. Actually all of these objections are unfounded. The good that is accomplished far surpasses petty opposition.

It should be appreciated that the desire to carry out mass surveys of the industrial worker is based upon the fact that industry possesses the organization and machinery to reveal the incidence of tuberculosis among the largest segment of our adult population. These studies are not made because industry *per se* is considered the cause of tuberculosis. The whole problem is integrated with economic factors—living conditions, housing congestion, nutrition, personal and community hygiene, financial insecurity, and so forth. Quite frequently the condition within the four walls of the plant is better than it is down the street in the workman's home.

However, industry has been stigmatized with the accusation that "it is dirty, grimy, and dusty" and, being so, it is supposed to be an incubator for tuberculosis. Possibly such thinking is an escape from the responsibility to provide our workmen with better homes to live in, better food to eat, and more hours free from mental and physical strain. It might be possible that it provides certain minds greater comfort to blame a gas, or dust, or humid atmosphere as the cause of a given case of tuberculosis rather than to place the responsibility where it belongs. This statement is not made in defense of industry but rather to emphasize the fact that the medical profession and social agencies should lend their greatest aid to eradicate the common, proved factors which encourage the development of tuberculosis.

The statement that an industrial environment does not produce or aggravate tuberculosis is not an idle, thoughtless generalization. A silica-laden atmosphere as well as a few other rare exigencies which might lower a workman's resistance are, of course, excluded from this discussion. The effect of certain occupational environments upon the production of tuberculosis has been given careful study over many years. It is a fact that nurses and medical students are liable to contact an open carrier of tuberculosis, and therefore their occupation constitutes a hazard peculiar to that employment. As to all other occupations, the evidence is, by and large, to the contrary.

At intervals, various medical organizations, especially those concerned with industrial health and such research institutions as the Trudeau School of Tuberculosis, have gathered experts in the field of tuberculosis and industry to present symposiums on the subject. The following opinions have been taken from the Trudeau Foundation Symposium.[1]

**Respiratory Irritants: Dusts, Fumes, Gases** *(Gardner[1])*.—The influence of fumes and gases upon susceptibility to tuberculosis is now receiving considerable attention. Upon the supposition that they are irritating and because some of them in large doses provoke intense inflammatory reactions, many physicians believe that these agents must affect associated tuberculosis of the lungs. On the influence of acute reaction to excessive dosage of gases, our best source of information is the experience of the World War. The Report of the Surgeon General of the U. S. Army for 1919 showed that among 70,552 American soldiers who had been gassed, 173 cases of tuberculosis subsequently developed, a rate of 2.45 per 1,000. The corresponding rate for all enlisted personnel overseas was 3.50 for 1918 and 4.30 for 1919. In 1930 Price summarized the experience for the armies of all the warring nations and reported the general feeling that war gases had no influence upon susceptibility to tuberculosis. This mature conclusion reversed that reached after the first year's experience, when most authorities were convinced that the acute inflammatory reactions must have been responsible for the increase in number of tuberculosis cases in the armed forces. Koontz made numerous experiments on the effect of gassing tuberculous rabbits for the Chemical Warfare Service and reported no alteration in native susceptibility to this infection.

The evidence on the influence of prolonged exposure to nontoxic concentrations of various gases is less complete. Evans reported his findings on annual roentgenographic examination of considerable groups of men engaged in the manufacture of chlorine, sulfur dioxide, hydrofluoric acid, and combinations of phosgene with phosphorus oxychloride and phosphorus trichloride. The atmospheric concentrations of these gases were not known but occasional accidents occurred and the hydrofluoric acid was particularly hard to confine. Enough escaped into the atmosphere so that men with glasses could not work in the rooms, and the windowpanes were etched to the extent that the wind blew them out of their frames. The films of these men, employed at such work for four to ten or more years, showed no trace of reaction attributable to gas, and the incidence of tuberculosis was no greater than in persons employed in other departments of this large plant. Repeated examinations failed to demonstrate evidence of reactivation of several cases of latent apical disease that occurred in the group.

Two old experiments are occasionally quoted to cast suspicion on sulfur dioxide as an excitant of tuberculosis, but this evidence is insufficient to incriminate it. In 1886 Celli and Guarnieri made four dogs inhale this gas in unspecified concentration and then had them inhale dried sputum. Three developed pulmonary tuberculosis, and as only one of seven controls became infected, he concluded that the gas had lowered susceptibility. Again in 1904 Kisskalt exposed a few tuberculous animals to uncontrolled amounts of $SO_2$ and found that the infection was aggravated, but his results have also been questioned.

In an analysis of the working environment and health of employees of nonferrous mines and metal smelters of the state of Utah, it was found that for the men in the smelters there was a tuberculosis rate of 5.4 per cent, whereas in the coal and metal mines the corresponding rates were 2.6 and 2.5 per cent, respectively. Most of the infection in the smelters was attributed to free silica, but a number of the men were not exposed to such dust. The possibility that $SO_2$ might have affected the picture was considered, but the evidence was not convincing. Kehoe, Machle, Kitzmiller, and LeBlanc reported on the examination of ninety-five persons employed in an electric refrigerator plant where sulfur dioxide was the refrigerant fluid. Most of these men had symptoms of upper respiratory irritation, but their lungs were quite free from disease and there was no evidence of tuberculosis in roentgenograms. Flury and Zernik reported that in Europe workers in sulfite cellulose factories and on producer ovens remain relatively free from all infections of the respiratory tract, including tuberculosis.

Finally, reference will be made to an uncompleted experiment upon the chronic effects of inhaling fumes generated in arc welding. The type of coated rod employed liberates fumes consisting chiefly of iron, silica, and manganese with no fluorides. The gas reaching the exposed animals consists largely of oxides and nitrogen. Both normal guinea pigs and those with primary tuberculosis infection produced by the inhalation of attenuated tubercle bacilli have now been exposed to sublethal concentrations of this gas-fume mixture for a period of four months. At first there were a few deaths from chemical pneumonia, but these promptly ceased on decreasing the concentrations slightly. As in all lungs irritated by the inhalation of both benign and dangerous dusts, the distribution of tubercles was abnormal and their number and size were appreciably greater than in the control series. Up to now, however, there has been no tendency for the infection to spread beyond the primary sites of localization. In the light of other experiments of this type it seems probable that no dissemination will occur and that the ultimate effect upon the tuberculous infection will be significant.

Sufficient evidence has been cited to indicate that the mere presence of inflammatory reaction in lung tissue does not alter native reaction to the tubercle bacillus by purely mechanical means. There is no proof that local inflammation favors the implantation of tubercle bacilli nor does it necessarily bring about dissemination of infection from a pre-established focus of tuberculosis. The significant factors responsible for the unfavorable course of a tuberculous process are more likely chemical in nature and only created by a limited number of specific irritants. In the category of such irritants we now recognize free silica tuberculin, and possibly the virus responsible for influenza. None of the evidence available at the present time indicates that dusts, other than free silica, either in pure state or as a contaminant of other materials, or any of the more common industrial fumes or gases have specific action in altering susceptibility to tuberculosis.

**Lead Absorption Versus Tuberculosis** *(Belnap[1]).*—In the course of my experience with over 500 cases of lead absorption in the past fifteen years, covering a wide variety of lead trades, I have had opportunity to observe possible effects of absorption and lead intoxication on the disease picture which we call tuberculosis. For the purpose of being very specific, my discussion is based on my own clinical studies for the past five to eleven years of a particular group of men exposed by inhalation to large concentrations of lead dust in the storage battery industry. This particular group of workers includes ninety-seven men ranging from 25 years to 59 years, with the majority 34 years or over. Seventy-seven of these men are still working in the same storage battery industry, while twenty have quit or been laid off. Seventy-two of these storage battery workers have worked five years or over and 49 per cent ten years or over. The following conclusions are reached after this experience in the lead industry.

1. Lead absorption, even if complicated by actual disabling lead intoxication such as lead colic, does not reduce general resistance to upper respiratory infection, or nonspecific pulmonary infections.

2. Lead absorption does not impair general health as shown by absence of weight loss and practically normal sedimentation throughout the series.

3. Lead absorption does not produce the fibrosis which can be demonstrated in the case of silica inhalation.

4. Lead absorption or intoxication does not predispose to or aggravate pulmonary tuberculosis.

**Temperature and Humidity Versus Tuberculosis** *(Baetjer[1])*.—From a survey of industry it is apparent that no statement can be made regarding the relation of temperature and humidity to tuberculosis. The value of such a discussion lies only in presenting the almost complete lack of knowledge on the subject and the great need for work in this field. Mortality statistics in industries having high temperatures and humidities indicate no very clear effect of temperature and humidity. Furthermore, such figures are open to severe criticism because of the impossibility of eliminating the many other factors involved. Climatic studies again have yielded very inconclusive evidence. Very little experimental work has been done in this field, but insofar as it goes it has been negative. From a consideration of the physiologic effects of temperature and humidity, it is difficult to see how temperature and humidity could markedly affect tuberculosis. In conclusion, one can only emphasize the incompleteness of our knowledge on this subject, and hope that further experimental work and statistical analyses will throw more light on the problem.

**Tuberculosis Among Steel Plant Workers** *(Kammer[1])*.—1. Analyses are made of records of 662 cases of tuberculosis discovered in routine examinations of 15,000 employees and applicants for work at a steel plant. Chest roentgenograms were made of all examinees, and diagnoses and opinions of progress of cases were based on roentgenographic findings. All cases of secondary, or adult type, infection are included in the study. Almost all of the cases were in males.

2. Indirect evidence is presented to show that hard physical work in hot environments does not predispose workers to pulmonary tuberculosis. An analysis of the work histories of the tuberculous examinees shows that they worked about ten years at their longest jobs and about five years at their second longest jobs regardless of the degree of physical effort required to do their jobs. Reference is made to a previous publication showing that when a suitable correction is made for mean ages in the two groups, the incidence of pulmonary tuberculosis is no greater among employees than among applicants for work at the plant.

3. An attempt is made to measure the progress of known cases of tuberculosis under varying degrees of physical effort at work and varying degrees of heat in environment at work places, and to determine what influence age and race had on the progress of the cases. All estimations of progress are based on dispositions made of cases by members of the clinic staff at the time of first examination when tuberculosis

was discovered or during the ensuing follow-up period. The method embodies the use of personal and subjective factors, *viz.*, judgment, and previously held opinions on the part of the medical staff, and varying degrees of desire and ability to cooperate on the part of the patients. It is believed the fault has been overcome by a proper use of the statistical material, and that the dispositions of cases over a prolonged period of time must summarize the minor trends of progress of the cases.

4. Tuberculosis among workers in their twenties was more frequently a serious clinical problem than was tuberculosis among workers at other age periods. The tables reveal that clinically significant tuberculosis, much of which needs to be treated, is to be discovered in workers in their fifties and sixties.

5. The incidence of pulmonary tuberculosis among steel plant employees was determined in a previous publication to be the same for the three races: white, Mexican, and Negro. The present report establishes that the cases of tuberculosis among Mexicans are more apt to be clinical problems than are the cases among white workers. No conclusion is drawn with respect to Negroes.

6. Work in hot environments did not have an unfavorable influence on the progress of the cases under study.

7. Tuberculous workers on light jobs managed their infections somewhat better than did those on hard or moderately hard jobs. The difference was not enough to warrant a feeling of security with respect to clinically significant tuberculosis among workers on light jobs. Once discovered, such cases require treatment with the same promptness as do cases in workers at physically hard jobs. It was not determined whether physical effort was an independently operating factor or a part of a complex with age and race.

**Tuberculosis in the Foundry Industry.**—The incidence of tuberculosis in the foundry industry has been studied by Greenburg[1] in New York state, Pope[1] in Massachusetts, and Sander[*] in Wisconsin, only to name a few. The evidence is that among those whose work does not expose them to silica, the incidence of tuberculosis has been little different from that among other industrial groups. The difficulty, however, is that there exists in most foundry work a certain amount of silicious dust, and, because of this, the tuberculosis rate among foundry workers is, as a whole, fairly high.

**Tuberculosis in Granite and Marble Workers** *(Benson[1])*.—Pulmonary tuberculosis, as has been shown in the past, is a definite hazard for the worker in granite dust, but does not appear to be an occupational hazard for the worker in marble dust.

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[*] Sander, O. A.: Tuberculosis and Its Relation to Silicosis, Indus. Med., 7: 309, 1938.

**Tuberculosis in Hard Rock Miners** *(Cummings[1]).*—1. Tuberculosis hazards in the mining industry usually are associated with the silica content of the rocks encountered.

2. Information on the nature of specific tuberculosis hazards in industry is reviewed by the author in another publication.

3. Recent studies concluded over a five-year period among a large group of white male employees in the mining industry suggest:

a. That among individuals with pulmonary infections, presumably tuberculous, the proportion of active lesions is greater in silicotics than in presilicotics or persons with no evidence of pulmonary fibrosis.

b. That among individuals with active or indeterminate lesions, presumably of tuberculous origin, the proportion which tend to become worse with the passage of time is greater in silicotics than in presilicotics or persons with no evidence of pulmonary fibrosis.

c. That among individuals with apparently healed lesions, presumably of tuberculous origin, the proportion which tends to become worse after a few years is so great in silicotics that there is little justification for considering any tuberculous lesion healed in the presence of silicosis.

d. That among individuals showing no evidence of pulmonary tuberculous infection, the chance of developing a lesion of this type appears to be twelve to twenty-four times as great in silicotics as in those with no evidence of occupational fibrosis.

e. That individuals may develop new lesions, presumably tuberculous in origin, at any age between twenty and seventy.

f. That silicotics show marked differences in their tendency to develop new pulmonary lesions depending upon the individual opportunities for contracting infection by contact with open cases of tuberculosis.

g. That the chance of dying from tuberculosis, always great for the silicotic, is profoundly influenced by the degree of contact with infecting organisms in his environment.

**Fatigue as a Factor in Industrial Tuberculosis** *(Wishard[1]).*—Considering occupational fatigue as a definite factor in activating a pre-existing tuberculous lesion or contributing to the spread of a new one, one should first have some definite concept as to what constitutes industrial fatigue. Florence defines it as a "diminution of working capacity caused by length or intensiity of activity in a gainful employment" and insists that one must first show a definite diminution in working capacity and associate this with a definite increase in the length or intensity of this activity. He then enumerates a large number of factors contributing to fatigue but makes no mention of personal or physical ones, such as chronic infections of various organs, endocrine unbalance, deformities whose presence necessitates an additional outlay of physical energy, and the like. Florence is not alone in his approach to this subject. Many writers have written at great length, listing such factors as the color of the walls, types of chairs, noises, etc., all of which doubtless do have a certain bearing on the development of fatigue, but certainly not as much as a subnormal physical state. It is our opinion that occupational fatigue may be considered as that diminution in physical effiiciency brought about by the expenditure of a given amount of physical energy or mental strain in the course of the performance of a day's work according to present-day industrial standards. If one could consider merely the energy loss incident to one's employment, the problem would be relatively simple, but that cannot be done. For many persons, the day does not start until after they have left their place of employment. Again, age does not appear to matter. While the younger individual may spend his evenings in a night club, the older person will spend an equivalent period in his basement carpenter shop or cultivating his farm, or the married woman will toil far into the night to complete the family washing and ironing. In this connection, an employee recently made a somewhat interesting observation. He stated that he felt better when working longer hours as he was so tired that he stayed at home in the evenings, whereas, formerly he had prowled about town until all hours. It would appear that a full forty-hour week or even a forty-five hour week is less strenuous on our employees than a relatively short one. Women with household duties are not so ready or willing to do them when they work longer hours and have more money available with which to employ some neighbor girl. Similarly, employees in general limit their social activities to the week end and in this manner they get more rest and feel better than during periods of less factory work.

It is generally recognized that *chronic infectious states* and *other pathologic conditions* will lower the fatigue threshold far below that obtainable by the *expenditure of muscular energy.* (Italics are the author's.) In fact, the theory has been advanced that the human mechanism cannot arrive at a stage of pathologic fatigue from muscular exertion alone—that the muscular system will refuse to respond further, long before this stage is reached. On the other hand, surveys of physical examination records of large numbers of factory workers show almost half to be in need of some sort of medical or surgical care. That this does not apply to factory workers alone was strikingly shown by the physical records of the men examined for military and naval service.

**Summary.**—At the close of the Symposium on Tuberculosis in Industry, held in 1941 (data published in 1942), Dr. Leroy Gardner stated that from the opinion offered by the various authorities, "we are agreed":

> That there can be no tuberculosis without tubercle bacilli;
>
> That the opportunities for infection vary widely in different communities and in different social strata in those communities;
>
> That the prevalence of disease is not uniform;
>
> That the source of infection is invariably another tuberculous individual;
>
> That transmission of infection is usually in the immediate vicinity of the open carrier;
>
> That most clinically significant tuberculosis is the result of reinfection although we recognize that sometimes primary tuberculosis may go on to give rise to such disease.
>
> As to when and how infection or reinfection occurs, there has been difference of opinion. Presumably the incubation period is measured in months or even longer periods.
>
> All the surveys reported indicate a rising incidence of tuberculosis with advancing age. The bulk of new cases were found between ages 15 and 30, a small number of cases developed between 30 to 50. Some of us have interpreted the latter as carry-overs in resistant individuals from earlier periods when tuberculous infection was more general than today.
>
> Serial examinations in three large groups demonstrated at *all* ages new evidence of tuberculosis where none was seen before. For normal people this occurred at a rate of one case per 2000 per year.
>
> The obvious deduction is new exogenous infection followed by disease in a short time.
>
> The likelihood that apparently healed tuberculosis might break down and spread after many years has been debated. Most have felt that it would not, except when subjected to the action of free silica dust.
>
> The experience of a few was to the contrary, particularly with radical changes in environment.
>
> The occupation of attending to the sick may involve unusual opportunities for infection, and under these conditions we must consider tuberculosis as an occupational disease.
>
> Of the environmental factors, that all-embracive one, designated as the *standard of living,* is universally accepted as significant.
>
> *Nutrition,* a component element, has long been recognized as fundamental. Precise measurement of its influence is afforded by variations of the Ca and N balances in growing children.
>
> Of all the accessory environmental factors peculiar to industry only free silica is accepted without challenge.
>
> Exposure to *fumes and gases* coud not be proved to favor the development of tuberculosis.
>
> *Lead absorption and intoxication* were not associated with any excess of tuberculosis in one careful study.
>
> *High temperature and humidity* were without significant influence upon the tuberculosis rate in exposed workmen, nor were there theoretical reasons to suspect they should.

*Radiant heat* in the steel industry caused no excess tuberculosis in those exposed.

*Fatigue* is recognized as unfavorable to the tuberculous sanatorium patient.

If the employees in the automotive or steel industries suffer fatigue it is not reflected in their tuberculosis rates.

*Trauma* may reactivate preexisting latent tuberculosis. Whether it favors localization of new infection is debatable. Often it presumably attracts attention to a pre-existing lesion.

In the absence of x-rays before and immediately after injury, proof of its influence, without specific symptoms, is difficult.

*Tobacco*—There is little excess tuberculosis in the modern cigar and tobacco factory.

Only 25 per cent of the labor in this industry works under such conditions, however.

Of the *dusts,* only those high in free $SiO_2$ are known to alter susceptibility to tuberculosis appreciably. Exposure to grain dusts in one group was associated with more tuberculosis than anticipated but socioeconomic factors could explain the excess.

Exposure to marble, gypsum, and cement dust have not resulted in striking increases in the tuberculosis rate.

New data on the incidence of tuberculosis in the foundrymen, hard rock miners, and granite cutters all reaffirm our belief in the effects of silica in these industries.

In all of them the incidence of tuberculosis is high, rising with the years of exposure and the amount of silicosis in the lung.

The infection may precede the silicosis, remain dormant for years, and finally reactivate to terminate fatally.

The infection may be superimposed and then run an acute or a chronic course, depending upon unidentified conditions.

Infection is not essential to the development of silicosis but when present in the healed or active stages it probably favors development of silicotic reaction.

The basis of control is education and prevention, and the essence of prevention is discovery of the case and its prompt treatment.

**Industrial Environment and Pneumonia.**—It has been pointed out in previous chapters that in certain circumstances a number of chemicals, when inhaled, can produce chemical pneumonia or pneumonitis as a primary effect. Other chemicals may cause such a severe inflammatory reaction in the lungs that pneumonia may result as a secondary complication. Except for those specifically mentioned, the gasses, fumes, vapors, and dusts of industry do not affect the lungs. In the majority they are inert, or pass through the lungs to produce systemic disturbance, such as carbon monoxide or lead.

Occasionally, "job requirements" subject men to unusual conditions which lower body resistance. Obvious examples of this type of exposure are policemen or firemen who, on a cold winter night, may become water soaked but must remain on the job. Pneumonia following such an exposure is certainly to be attributed to the occupation.

The vast majority of occupations, however, do not influence the development of pneumonia. Fact-finding surveys have been conducted in many industries to determine any relationship between occupation and pneumonia. When the weather factor has been ruled out, it appears that pneumonia is not influenced by the majority of occupations.[2]

## References

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2. Collen, M. F., Dybdahl, G. L., and O'Brien, G. F.: A Study of Pneumonia in the Shipbuilding Industry, J. Indust. Hyg. & Toxicol.. **26**: 1, 1944.

# CHAPTER XXXIV

# THE DERMATOSES

Adequate consideration of the occupational dermatoses requires a complete textbook, and no brief chapter such as this can hope to cover the subject satisfactorily. Furthermore, a discussion of the dermatoses is rightly the prerogative of the dermatologist. For that reason this chapter has been written largely by Dr. Nelson Paul Anderson, of Los Angeles, with but few additional observations by the author of the book. Certain lesions caused by metals, solvents, and plastics have been described in the discussion of those metals.

Too much emphasis cannot be made on the necessity of preventing occupational skin lesions. A good control program does not consist of a set of arbitrary rules and regulations which the employees may be requested to follow. The primary requisite regards the chemicals or other factors present in the working environment. The plant physician should design the preventive program according to the type of skin hazards existing in any given plant.

**Occupational Hazards.**—It is impossible to list the occupational hazards, for it may be truthfully said that the majority of all trades present such a hazard to certain persons. It is known that any substance encountered in any trade or occupation may produce a dermatitis in one who is susceptible. Occupations involving the handling of oils, chemicals, dyes, paints, solvents, metals, fabrics, rubber, and heavy dusts, cement in particular, lead the list.

**Definition.**—An industrial dermatitis is any inflammatory disease of the skin for which industrial exposure can be shown to be a major causal, contributory, or eliciting factor. Industrial dermatitis is a subcategory of occupational dermatitis, for occupational dermatitis includes not only industrial dermatitis, but also all other dermatitides attributable to any occupational pursuit.[2]

**General Considerations of Etiology.**—That allergy plays a role in the production of many types of occupational dermatoses seems to be an established fact. In spite of the tremendous amount of investigation on this subject, little is known regarding the true mechanism of the production of an allergic state. For practical purposes the physician handling occupational dermatoses should realize that allergy and hypersensitivity do exist. He should, however, fully realize that there are many other factors which may play an important part in the causation of dermatitis in industry and that these factors may or may not deal directly or indirectly with allergy.

Many of the skin conditions met with are due to primary cutaneous irritants. These substances, which are generally strong acids, alkalis, or corrosive salts, are sufficiently irritating to produce a dermatitis in a large percentage of persons with normal skin.

The presence of focal infections in the teeth, tonsils, and sinuses undoubtedly contributes to the production of a dermatitis in certain cases. Superficial fungous infections of the feet and groin give rise to many perplexing problems in industrial dermatitis. The type of skin, perspiration, diet, and personal cleanliness are all factors which must be taken into consideration.

Finally, as pointed out by Sulzberger and Finnerud,[2] there are many eruptions which are due to an interaction of many elements and to a complex effect attributable to the cooperation of many factors. He terms this action "synergic" or "synergistic."

**Diagnosis.**—Certain lesions resulting from occupational exposure constantly present the same clinical manifestations. "Chrome holes" on the hand, or of the nasal septum, have constant characteristics which should entail no diagnostic difficulty when appearing in a man working with these fumes or with the chromates. The same may be said of the furunculosis occurring in oil or cement workers, due to mechanical blockage of the pores of the skin, or the acne from the action of tar. For these and many others, the industrial cause is usually apparent. The industrial physician, practicing in an area given to the manufacturing or processing of certain substances, rapidly learns the characteristics to be expected in lesions likely to result from such exposure.

On the other hand, there are vast numbers of what appear to be nondescript eczematous lesions of occupational origin which are most baffling. With these it is not such a simple matter to find the causative agent. Furthermore, these lesions do not take on a characteristic appearance such as the chrome ulcer, but instead present an eczematous appearance similar to many other dermatoses. A dermatitis from a plant, a dye, a solution, a cosmetic, or a fungus may be clinically indistinguishable by the physician. It is this type of case which must frequently be referred to the dermatologist.

History.—A careful and detailed history should be taken before proceeding with the examination of the patient. This history must include an inquiry into all past illnesses of the patient and his family, with particular reference to any previous skin trouble, asthma, hay fever, urticaria, or other allergic disorders, and of any drugs recently taken. Detailed chronologic statements should be obtained, concerning both the particular type of work done by the patient and the onset of the present eruption. Inquiry can be made about the working conditions, including light, heat, ventilation, and factory hygiene. One should try to obtain a complete and vivid picture of the patient's daily routine while at work. While doing this, a list can be made of substances, chemicals, or other materials which the patient contacts while at work. The history is completed with information regarding the patient's outside activities, conditions of home life, and hobbies.

Removal of Clothing.—There is one admonition which cannot be too strongly stressed. Any patient presenting a skin lesion should be examined with all clothing removed. A Colles fracture may be examined and diagnosed with only the forearm bared, but a skin lesion of the wrist requires removal of the entire clothing for adequate diagnosis.

Criteria for Diagnosis.—In order to establish proof of the industrial nature of a given dermatitis, Sulzberger and Finnerud[2] listed certain criteria which should be of aid. These are quoted as follows, except that under their Criteria of the Second Order I have considerably altered their seventh point.

### *Criteria of the First Order*

1. Inception.—The dermatitis appears at any time during a period of industrial exposure or even after a lapse of a reasonable incubation period following the cessation of the industrial exposure (usually a maximum of from two to three weeks).
2. Amelioration.—The dermatitis regularly disappears or is repeatedly improved within a reasonable period (days, weeks, or even months) after cessation of the causal industrial exposure. (While this is usually the case, retention of causal agents, complications, or ensuing polyvalent sensitization may prolong the course even for several years after the last industrial exposure.)
3. Recurrences and Exacerbations.—The dermatitis shows a tendency repeatedly to recur or to exacerbate when the worker returns to the identical industrial exposure after a certain period of absence (provided that there has been no change in working conditions or in the patient's manner of working, or in his susceptibility).

### *Criteria of the Second Order (Adjuvant Criteria)*

1. The dermatitis appears first in, and is usually confined to, the areas of maximum exposure (in a small percentage of cases it spreads to, or may begin in, apparently unexposed areas or even becomes generalized).
2. The character and localization of the dermatitis correspond to the character and localization of dermatitis known in other cases to have been caused by exposure to the same or similar industrial hazards. (While many different substances and procedures can produce similar or identical eruptions, there are certain classes of substances and of procedures which regularly produce fairly characteristic lesions.)
3. The application of the presumptive causal agents to an unaffected site close to the site of the dermatitis produces a reaction, provided this application is made either during the active phase or after the proper interval following the cessation of the dermatitis.
4. The cutaneous test produces reactions of the same fundamental nature as the dermatosis under investigation.
5. Other workers similarly occupied are or have been similarly affected.

6. The dermatitis appears soon (days or weeks) after the patient begins work involving new potential hazards.
7. The dermatitis is proved to be of possible occupational nature; that is, it is shown to be of the type which may result from the industrial exposure sustained.

SKIN DISORDERS UNRELATED TO OCCUPATION.—The physician should recognize that such skin disorders as psoriasis, scabies, pityriasis rosea, seborrheic dermatitis, impetigo, erythema multiforme, and lichen planus are practically never related to occupation. Nevertheless, patients with these diseases frequently present themselves, believing the skin disorder to be caused by their work. It occasionally happens that a pre-existing skin condition, particularly seborrheic dermatitis, may be a predisposing factor in the production of an industrial dermatitis.

PATCH TEST TECHNIQUE.—The patch test is performed as follows: The substance to be tested is placed upon a small square of gauze, several layers in thickness. This gauze square is most convenient when about three-fourths the size of a postage stamp. In general, powders or dry substances must be moistened slightly with water. This small patch is then placed upon the skin in such a manner that the substance to be tested comes in direct contact with the skin. Any area of the skin may be selected as the test site. The areas preferred are the back, inner aspects of the upper arms, and the anterior surfaces of the thighs. In industrial practice, particularly if one has any suspicions of the integrity of the patient, the back is the best location for testing. The patch is then covered by a square of impervious material, such as wax paper or cellophane. This covering should be about four times the size of the test patch. This is covered by wide strips of adhesive and left undisturbed for forty-eight hours. The patient should be instructed to remove the patch at once if burning, stinging, or marked discomfort occurs. In cases of known sensitivity to adhesive tape the patches may be held in place by flexible collodion, nail polish, or rubber cement.

*Delayed Reaction.*—After removing the patches, the test areas may be encircled with 2 per cent mercurochrome or 2 per cent gentian violet, in order to facilitate the correct reading of delayed reactions. The reading of the test is performed twelve to twenty minutes after removal of the patches and should be checked by subsequent readings at forty-eight and ninety-six hour intervals.

*Characteristics of Positive Reaction.*—A positive reaction is characterized by a sharply defined square of redness, corresponding in size and shape to the central test area (Fig. 66). Papules and vesicles are also usually present, corresponding to the appearance of the original eruption. Only rarely is a positive reaction manifested by redness and inflammatory edema. Such a positive reaction can best be determined by feeling or stroking the test area with the finger.

*Evaluation of Negative Reactions.*—Having performed patch tests, one would think it a simple problem to evaluate the results of such tests. In the ordinary case this is true. There are, however, numerous pitfalls in the interpretation of such tests. Negative reaction to patch tests with suspected irritants can present several sources of error:

1. The dermatitis in question may not be the result of any substances contacted at work.

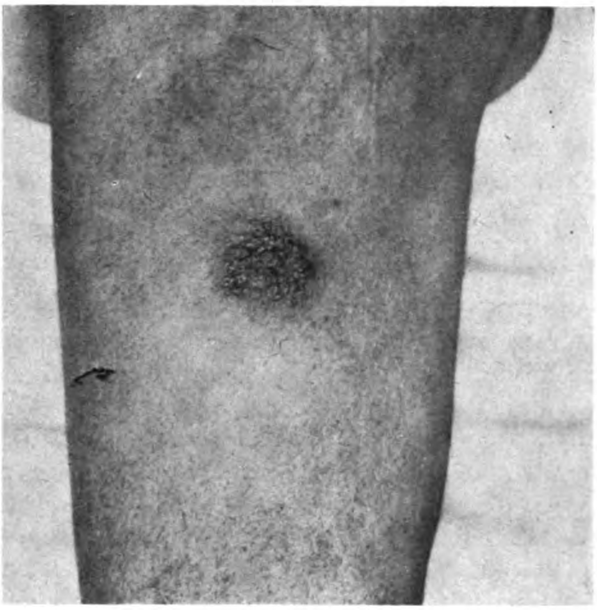

Fig. 66.—Positive reaction to patch test from 1 per cent formaldehyde.

2. Local skin sensitivity may be present. This phenomenon is rare, but it occasionally happens that patch tests on sound skin give negative results. In such cases the test may be repeated, using a site adjacent to that of the original eruption. It may be necessary to wait until the original condition has disappeared and then to repeat the test on the healed area.[3]

3. The actual causative substance producing the eruption may not have been tested; or, if tested, it may have been used in too weak a concentration, or incorporated in an unsuitable vehicle.

4. Theoretically possible, but actually quite rare, is the occurrence of a refractory period of skin reactivity. During such a time the skin may fail to react to a definitely causative substance. If the patch test is performed during such a refractory period, one can easily see how erroneous conclusions can be drawn.

5. The actual conditions of industrial exposure are never duplicated by patch tests. It is well known that the noxious effects of many substances are magnified by numerous external factors. These include perspiration, heat, cold, sunlight, repeated cleansing with soap and water, repeated exposures, and prolonged immersion in liquids leading to maceration of tissues. None of these contributory factors can be adequately reproduced by the patch test.

6. Occasionally, patch tests done with all suspected irritants are negative in spite of fairly definite clinical evidence that some industrial contact is causative. In such cases there seems to be definite evidence to show that two or more substances act in combination or synergistically to produce the occupational dermatitis.[4] This is particularly true in the so-called "baker's dermatitis." In other instances, in addition to exposure to some irritant, an additional physical agent, such as sunlight or heat, is necessary to produce a positive reaction.

*Evaluation of Positive Reactions.*—Positive reactions to patch tests with suspected irritants can present several sources of error:

1. Either substance tested may be a primary irritant which would produce irritation of any normal skin; or, if not a primary irritant, it may have been used in too strong a concentration in performing the test. Under no condition should the concentration employed in testing exceed that which occurs in exposure at work. The use of solutions in too strong concentration may give rise to localized gangrene and widespread irritation, and may even be responsible for the generalization of the heretofore localized process.[5] In fact, there are medicolegal possibilities in using the patch test. Certain chemicals must be incorporated into vehicles before testing. The proper vehicle and the concentration to employ have been made the subject of considerable study.[6]

2. A positive patch test with a substance contacted at work does not necessarily prove that the eruption was caused by this substance. Such a positive test may indicate only a latent sensitivity, and the real cause may be some substance not tested but contacted by the patient outside of his work.

3. The interpretation of the positive patch test is always open to the criticism that such a test can never actually duplicate the conditions of industrial exposure.

4. Marked difficulties of interpretation arise in cases where several different substances produce positive reactions to patch tests. This occurrence of polyvalent sensitivity, however, may furnish a clue to a nonindustrial irritant and at the same time aid the physician in avoiding contact dermatitis as a result of his own therapy. Of course, a patient found to have a positive reaction to mercury among other things would not be treated by mercurial ointments or antiseptics.

5. For a substance to be incriminated as the cause of an eruption there must be a definite history of contact with the suspected material.[7]

6. Although stated elsewhere, it may be said that with one or two exceptions (cement), the elimination of contact with the suspected irritant should produce either a cure or marked amelioration of the skin condition.

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In the following paragraphs consideration is given to a few representative and fairly common types of occupational dermatitis.

**Epidermophytosis.**—The relationship of superficial fungous infections or eczematoid ringworm of the extremities to industrial dermatitis is one that gives rise to more discussion and dissension than occur perhaps in any other skin disorder. As Blaisdell[8] states: "Broadly speaking, it would apparently be easy enough to discriminate between the case entitled to compensation and the one that is not, but considering the cases individually it is oftentimes difficult to give a judgment that is fair to both the worker and the insurance company."

There are definite instances where epidermophytosis might be directly related to the occupation; for example, an attendant in Turkish baths. There are also cases where a true contact dermatitis of industrial nature may be followed by secondary infection with ringworm organisms. Again, a contact dermatitis may be superimposed on a superficial fungous infection. This not infrequently happens in cases of cement dermatitis.

In every case of suspected industrial dermatitis of the hands, the experienced clinician will examine the feet for evidence of any superficial fungous infection. Such examination should include microscopic study of scales for mycelia. There is considerable clinical evidence to indicate that some of the eruptions on the hands in such cases are allergic manifestations of the fungous infection on the feet,[9,10] for example, epidermophytosis, and hence not of an industrial nature. However, in the presence of a positive patch test to some suspected and contacted irritant, such an infection of the feet may be looked upon as producing a nonspecific irritability of the hands. In other words, such a fungous infection of the feet lowers the threshold of sensitivity of the hands and hence acts as a predisposing etiologic agent.

The trichophyton intradermal test is believed to be of value if properly used. Lewis and associates[11] are of the opinion that while a positive intradermal trichophyton reaction is of little or no significance, a negative trichophyton reaction at least excludes the possibility of a given skin disorder's being an epidermophytid.

**Infectious Eczematoid Dermatitis.**—Traumatic wounds of all types are extremely liable to the development of infectious eczematoid dermatitis (Fig. 67), often erroneously considered to be a ringworm[12] infection because of its tendency to annular configuration. The usual picture is one of a person who sustains an injury to an extremity in the form of a scratch, abrasion, laceration, or crushing injury. In the latter event an amputation of one or several digits follows. During the period of healing an acute eczematous dermatitis develops about the wound margins. This is characterized by an annular or circinate redness with the presence of small pinhead-sized papulovesicles, producing a weeping surface. The condition may spread only in the neighborhood of the wound, but often patches are found far removed from the original site.

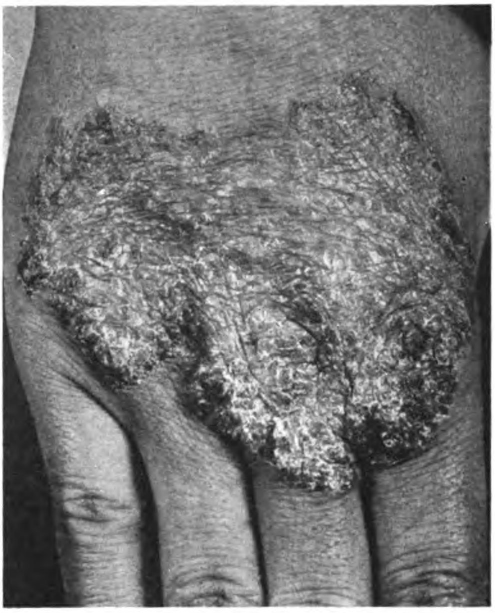

Fig. 67.—Infectious eczematoid dermatitis of the hand simulating a superficial fungous infection.

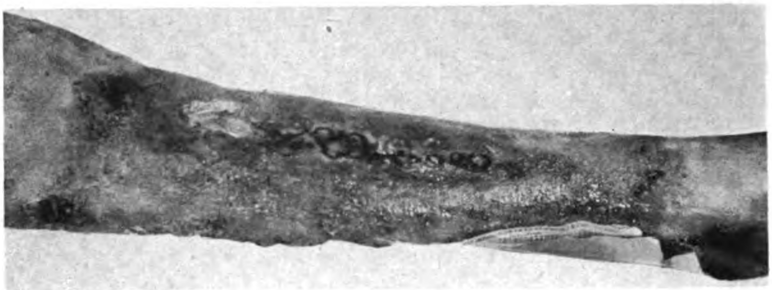

Fig. 68.—Diffuse infectious eczematoid dermatitis arising from multiple draining osteomyelitic foci.

Severe itching occurs, and the scratching which this causes may give rise to new lesions. It may also give rise to secondary infection in the form of lymphangitis, cellulitis, and regional adenitis.

A similar eruption may develop about draining sinuses subsequent to osteomyelitis and compound fractures (Fig. 68). Occasionally such an eruption is a true ringworm infection. In other instances the dermatitis may be due to sensitivity to mercurial or other antiseptics which have been applied.

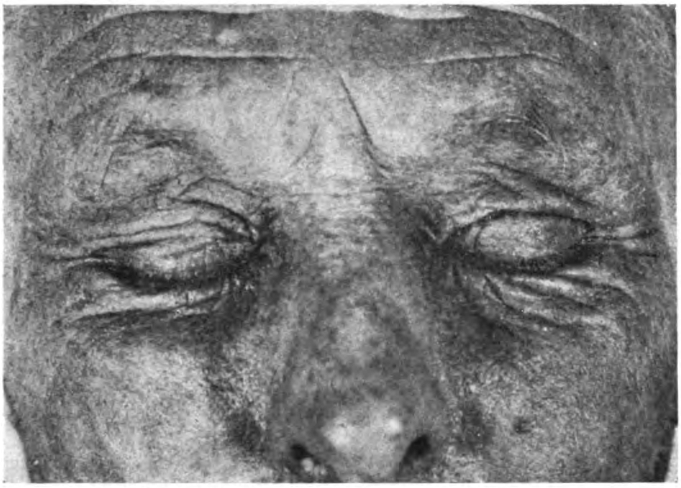

**Fig. 69.**—Contact dermatitis of the eyelids due to varnish remover. Such involvement of the eyelids may occur through fumes but is usually from actual contact through the medium of the hands.

**Turpentine Dermatitis.**—Turpentine is a frequent cause of contact dermatitis in industry. According to Schwartz and Tulipan,[1] it produces about 1 per cent of all cases of occupational dermatitis. Turpentine dermatitis may occur in those engaged in its manufacture; however, it is most frequently seen in painters (Fig. 69). Since it usually develops in people who have followed the painting trade for many years, it may present a serious problem both to the patient and to the insurance carrier. To the painter, turpentine dermatitis represents a serious obstacle to the pursuance of his trade. To the insurance carrier it represents a compensable occupational dermatitis in which the initial temporary disability is often a matter of several weeks and recurrence is practically universal.

There is no doubt that different kinds of turpentine vary a great deal in their irritating and sensitizing properties. Destructively distilled wood turpentine appears to be the least irritating.

**Baker's Dermatitis.**—Dermatitis in bakers is not uncommon and may result from contact with flour, sugar, cake dough,[13] bread dough,[13] cinnamon,[14] vanilla, various spices, and so-called "flour improvers," chiefly ammonium persulfate. However, only about two-thirds of the patients present positive reaction to ordinary patch tests. In such cases one must perform additional patch tests, using different combinations of the ingredients encountered at work. Even then there remains a considerable percentage of cases of unexplained origin.

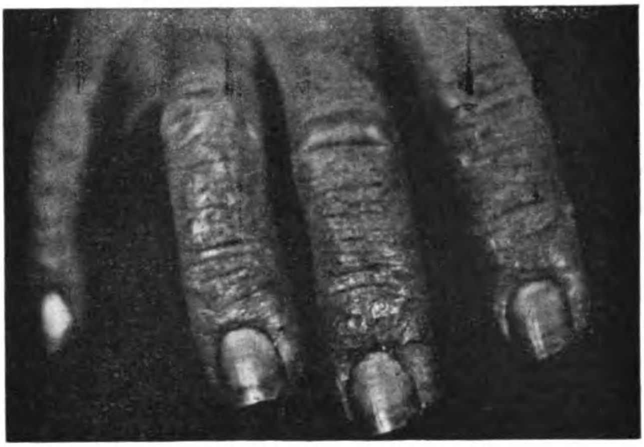

Fig. 70.—Dry, scaly, hyperkeratotic dermatitis occurring in a baker who was proved sensitive to both cinnamon and nickel.

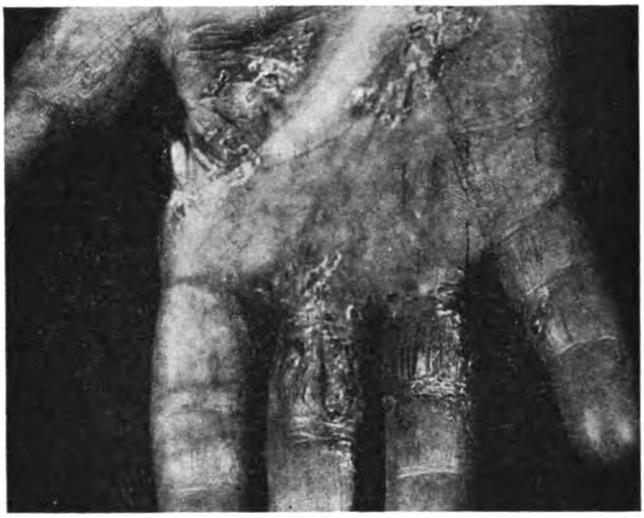

Fig. 71.—Occupational dermatitis of the hand, due to cinnamon, in a pastry worker.

The eruption involves the hands (Fig. 70), at times the forearms, and occasionally the face. It is usually of a dry, scaly eczematous[15] nature. In general, the eruption tends to be diffuse, and secondary ridging of the fingernails occurs in long-standing cases. Involvement of the nails alone may occur.[16] There are no other special clinical characteristics (Fig. 71).

**Cosmetician's and Barber's Dermatitis.**—Occupational dermatitis is common in those engaged in beauty shop work. Barbers are prone to develop a trade dermatitis from quinine, which occurs in many scalp tonics, from the perfumes used in many hair lotions, and from prolonged and repeated immersion of the hands in soap and water.

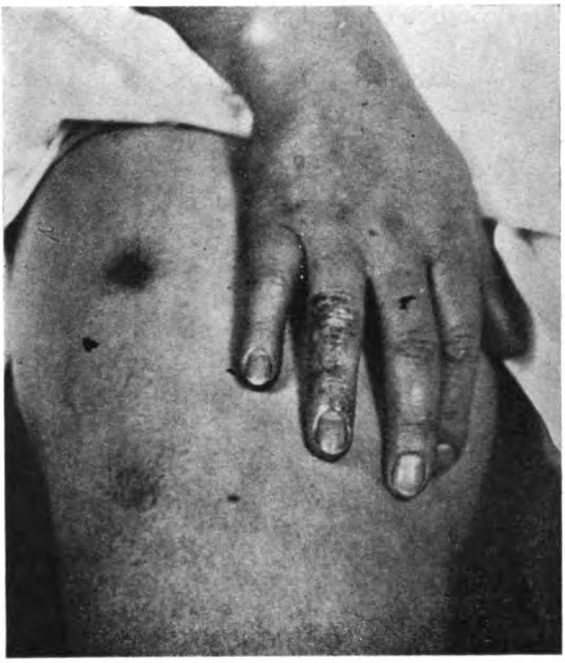

Fig. 72.—Occupational dermatitis in a hairdresser. Contact dermatitis of hand due to *p*-phenylenediamine; positive patch tests to same on thigh.

Cosmeticians handle many chemicals which may produce a contact dermatitis. These include p-phenylenediamine (Fig. 72), which is the active ingredient of most all hair dyes, nail polish, nail polish removers, finger-wave solutions, perfumed creams, rinses of all types, shampoos containing various oils; and, above all, formaldehyde, which is used frequently for the dry sterilization of combs and brushes. Regardless of other factors, it is important to find out the exact cause of the eruption in any given case. Once this has been determined, it is often possible for the beauty operator to continue in the same line of work, provided there is no exposure to or contact with the offending substance. The patch test with formaldehyde should be done with a 1 per cent solution.

**Cement Dermatitis.**—Dermatitis from cement is exceedingly common in masons, bricklayers, plasterers, and tile setters (Fig. 73). It differs from the ordinary contact dermatitis in that the eruption not only involves the exposed areas of the hands and forearms, but also occurs about the ankles and other covered parts of the body. Its persistence for weeks and even months after exposure has ceased and the tendency to secondary pyogenic infection are noteworthy. It is one of the most disabling of all the industrial dermatoses[17] and from many standpoints deserves more consideration from the dermatologist,[18] cement manufacturers, and insurance carriers than it has received. It should be noted that these patients may be sensitive to only one type of cement[19] and yet be able to handle other cements.

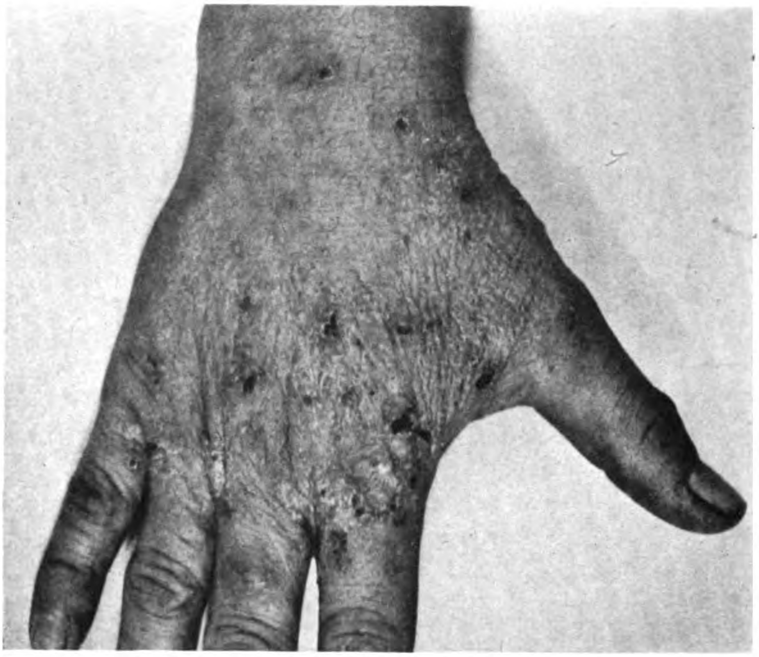

Fig. 73.—Contact dermatitis of the hand due to cement.

**Dermatitis Artefacta.**—One of the most difficult dermatologic problems confronting the physician is that of self-produced eruptions. Usually occurring in emotionally unstable but nevertheless intelligent persons, such artificially produced lesions have the following characteristics[20]:

1. Conform to no other known type of skin condition.
2. Occur in circumscribed areas, the surrounding skin being normal.
3. Can usually be reached by the hands and are often unilateral, being on the left side of the body in right-handed persons, and vice versa.
4. Other features of hysteria, for example, absent pharyngeal and conjunctival reflexes, may be found.

To the above may be added the observations that practically every patient has suffered some sort of trauma before the appearance of the first feigned lesion; and also that the first manifestation is often in the immediate neighborhood of the preceding injury.

Such feigned lesions are most frequently produced by acids, alkalis, caustics, or vesicants. They are usually ulcerative in character but may be erythematous, papulovesicular, or bullous. Gangrenous[21] lesions on apparently intact skin should immediately arouse the suspicions of the physician.

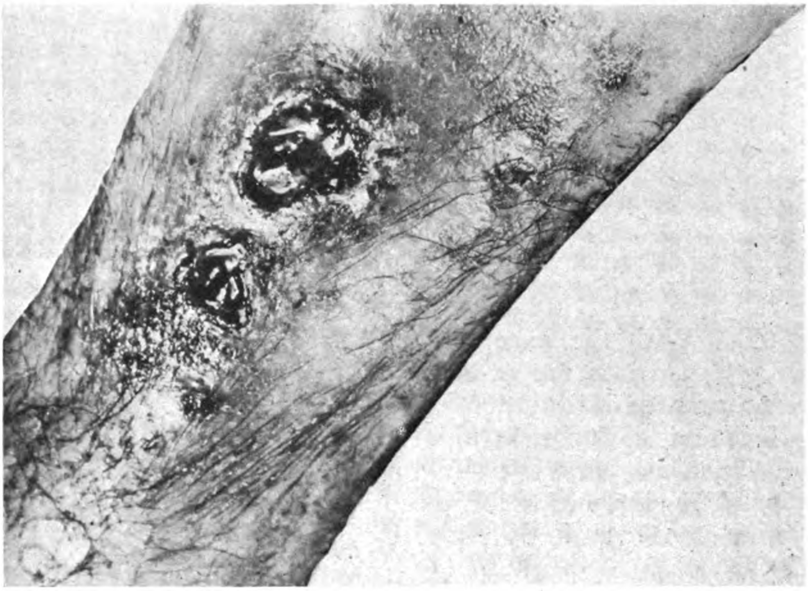

Fig. 74.—Dermatitis artefacta. Patient's first lesion was due to sulfuric acid from a storage battery. He subsequently intentionally produced the lesions seen in this picture in order to prolong compensation and avoid alimony payments.

Frequently in industrial practice, dermatitis artefacta is produced from self-evident motives, for example, compensation. At times other considerations come into play, including revenge or domestic difficulties (Fig. 74).

From a therapeutic standpoint, nothing is ordinarily gained by directly accusing the suspected person. Time and money will be saved by hospitalization for the use of occlusive dressings sealed with collodion or adhesive tape. If the latter is used, it should be marked in such a way that tampering with the dressing may be readily detected. Continuous strict observation must be carried out day and night, but in such a manner that the malingerer does not realize that he is under suspicion.

**Onychia.**—Involvement of the fingernails and paronychial tissues occurs more frequently in industry than generally realized.[22] Deformity, ridging, and transverse striations of the nails occur especially in soda fountain workers, cannery workers, fruit-juice stand employees, and others who have their hands immersed in liquids over a long period of time (Fig. 75).

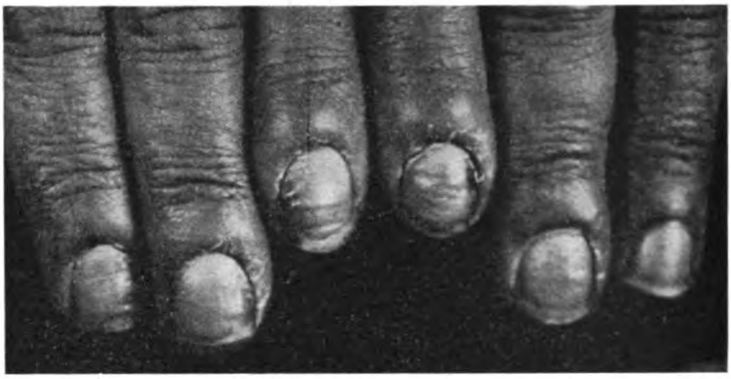

Fig. 75.—Paronychia with multiple involvement of the periungual tissues and secondary nutritional deformities of the nails, a common condition in soda fountain workers.

**Syphilis.**—With the exception of doctors, nurses, and laboratory workers, syphilis does not ordinarily enter into the question of compensable dermatoses. Situations do occur in which trauma acts as a contributory factor. The most important of these conditions is interstitial keratitis, which may first appear in a congenital spyhilitic following an eye injury. The criteria for the acceptance of such a diagnosis are well defined and have been aptly stated by Barkan[23]:

> "1. Trauma must be positively ascertained and more than such as the person is exposed to in the course of his employment.
>
> "2. Trauma must be of the cornea itself and cause a definite irritation.
>
> "3. An expert must determine that the interstitial keratitis is not present at the time of injury, and that the consequence of the injury is an interstitial keratitis."

Unilateral tertiary squamous syphilides of the palm occasionally appear to be induced by frequent and repeated minor traumatisms. Such a syphilide must appear on the right hand in a right-handed person, and vice versa. Further, the location of the syphilide must correspond to the area traumatized.

Finally, annular and circinate nodular and nodulo-ulcerative syphilitic gummas may appear in any area after trauma or after the removal of a foreign body. In general, compensability in all cases complicated by syphilis lasts only until the disappearance of the external manifestations of the disease.

**Medicolegal Aspects.**—While the industrial dermatoses are probably the most frequent of all industrial claims for compensation, the hearings of these cases before industrial accident commissions are infrequent. The insurer is usually willing to accept the diagnosis from a trained dermatologist. Less than 2 per cent of the cases handled at this clinic have ever been contested. Insurance companies also appear to be more willing to accept the skin lesion, since there is not the fear that permanent disability will face them if they accept the case as compensable.

TEMPORARY DISABILITY.—This ceases with the disappearance of the lesion, when the lesion does not interfere with ability to work, or when employment can be offered which is considered not likely to aggravate or produce further lesions. In some instances, loss of a wage difference between former occupation and the offered one has to be compensated.

PERMANENT DISABILITY.—This is rare. Occasionally, limitation of motion follows contraction scars or keloids from skin lesions. If so, they should receive a rating based upon the percentage of loss of function of the part. Disfigurement of the body as a result of alteration of the former appearance of the skin is ordinarily considered as not compensable.

**Treatment.**—

PROPHYLAXIS.—Preventive measures are highly important. Pre-employment examinations should uncover the history of repeated skin lesions or the tendency to react to even mild irritants. In occupations which involve a special liability to dermatitis the employer would do well to insist on a routine examination of the skin of prospective employees.[24] Such an examination should take into consideration the importance of any existing skin condition. Special emphasis should be placed on seborrhea, ichthyosis, hyperhidrosis, the general type of complexion (blonds and red-haired persons are very susceptible to cutaneous irritants), and the presence of epidermophytosis of the feet.

*Protective Procedures.*—Each new workman should be given careful instruction as to maintaining good hygiene, avoidance of undue exposure, and the wearing of certain protective devices. Wherever dust, grease, oil, grime, and the like are prevalent in the work (Figs. 76 and 77), the men should have two sets of lockers—one for the clean street clothes and the other for the working apparel. At the end of the day's work, he should deposit his working clothes in one locker, shower well, and then proceed to the second locker for his clean clothes. Men working where the substance is such that it may easily penetrate the clothing, such as many dusts, cement, or oils, should wear thick, closely woven clothes or rubberized material. Properly fitting gloves are often neglected where they would be advantageous. A film of oil or Vaseline may be a protection to unprotected parts. The value of this is questioned by most dermatologists, however. It is astonishing how many small plants fail to supply the aforementioned protective devices. Immersion of the hands in irritating chemicals is often done without gloves, when these could be easily employed. It can be laid down as a general rule that in all industries involving exposure to chemicals, dyes, drugs, coloring materials, or fluids, it is always advisable to rinse the hands or exposed parts with a weak solution of some reducing agent. If the offending agent is alkaline, then a weak neutralizing solution of vinegar, acetic acid, lime water, or weak sulfuric acid may be employed. For general purposes, especially

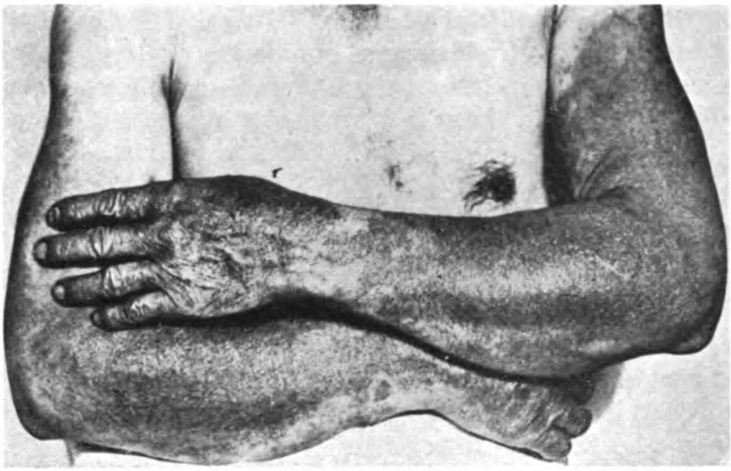

Fig. 76.—Contact dermatitis of forearms and arms in a janitor due to "sweeping" compound.

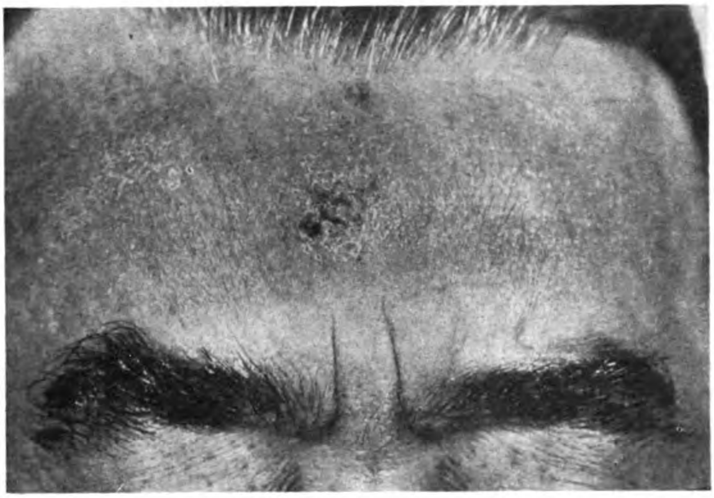

Fig. 77.—Occupational dermatitis due to leather. This hatband dermatitis caused by safety helmet in an oil field worker.

where acid is likely to affect the skin, a weak alkaline reducer may be used, as, for instance, a weak solution of sulfate of soda. These reducing agents should be supplied and should form a routine part of the workman's hygiene. After the use of a reducing agent, the parts may be washed in distilled water.

*Proper Use of Cleansing Agents.*—It is no exaggeration to state that a very considerable percentage of cases of so-called "industrial dermatitis" is due to the improper use of cleansing agents rather than to the actual contact of some specific industrial irritant. This entire field of industrial dermatitis has been extensively studied by Klauder and collaborators. The physician encountering negative reactions to patch tests in apparent and obvious cases of occupational dermatitis should take cognizance of this broad field. In probably 10 to 15 per cent of cases of proved contact etiology, soap, water, and other cleansing agents act as a definite contributory factor. The use of scouring or grit soaps, soap powders, washing powders, and laundry soaps is often followed by the onset of a dermatitis.

*Substitute for Abrasive Soaps.*—The proper cleansing agent for any particular plant or branch of industry can only be determined after proper study. As an efficient substitute for the popular mechanic abrasive soaps, Klauder and co-workers[26] recommend the following:

| Formula 12: | cc. |
|---|---|
| Sulfonated neat's foot oil | 45.0 |
| Light liquid petrolatum | 25.0 |
| Gelatin, 25 per cent aqueous solution | 10.0 |

This mixture is added to granulated corn meal in the proportion of 1½ parts by weight of corn meal, and one part by weight of the oil mixture. To prevent the growth of mold, a 0.5 per cent solution of chlorobutanol is added.

*Protection Against Chrome Compounds.*—In industries where chromic acid and chrome compounds are encountered (Fig. 78), that is, in dyeing, tanning, electroplating, and blueprinting,[25] a 10 to 30 per cent solution of sodium bisulfite ($NaHSO_3$) may be used as a reducing agent. It can be applied before exposure and allowed to dry on the skin and is also used as a wash after exposure.

Protective creams, liquids, ointments, and pastes may also be employed, their formulas and composition depending upon the requirements in each case. Klauder and co-workers[26] give a number of such formulas applicable to protection against prolonged contact with soapy water, or against dust-borne irritants. The industrial physician confronted with such a problem would do well to consult their article.

*Changing Working Conditions.*—Confronted with a severe and undoubted case of industrial dermatitis (Figs. 78, 79, and 80), there are two possible courses open. The first is to transfer the affected patient to another job, allowing most of the etiologic factors to remain for the next susceptible person. The second and more important procedure, particularly if numerous cases occur from the same source, is to change either the process or the working conditions. This involves an exact knowledge of all the processes involved. Such steps at times are made only when either governmental authorities or the insurance carrier exerts pressure on the insured.

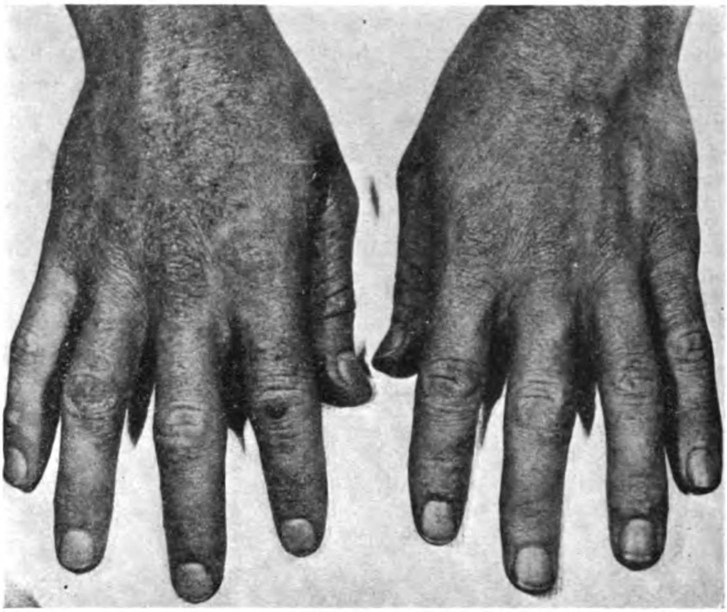

Fig. 78.—Contact dermatitis in a printer. Patient was sensitive to the bichromates used in this industry.

In most instances when a person severly afflicted with an occupational dermatitis changes his work, the skin condition clears and remains well. However, the skin condition may persist. Even if one continues at the same line of work, a certain percentage will eventually clear, although at least an equal number will continue to have skin trouble.[27]

Local Measures.—In the actual therapy of industrial dermatitis, certain local measures should be mentioned. If the dermatitis is acute and accompanied by vesicles and bullae, together with weeping and exudation, cold wet compresses are advised.

Liquor aluminum acetate (liquor burrowi, N. F.) . . . . . 480 cc.

Sig.: Dilute twelve to sixteen times with cold tap water and use as a continuous moist compress. Such compresses should be changed or remoistened every two to three hours.

If the process is acute and oozing but accompanied by any evidence of pus formation, the use of wet dressings of from 0.25 to 0.5 per cent of an aqueous solution of silver nitrate is indicated. Wet dressings of Alibour water (see Infectious Eczematoid Dermatitis) are also of value.

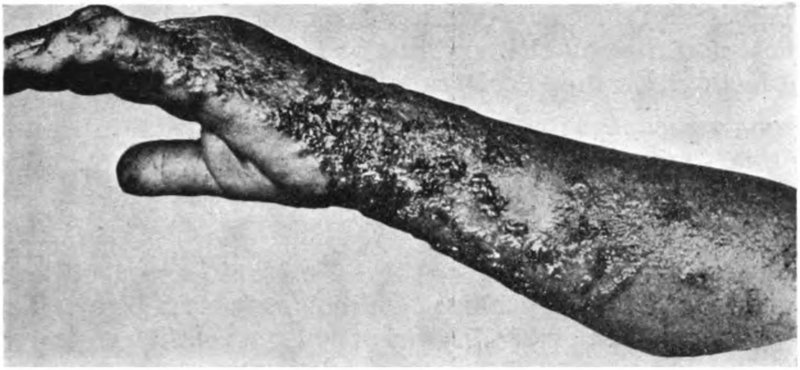

Fig. 79.—Severe bullous contact dermatitis of the forearms in a construction worker due to poison oak.

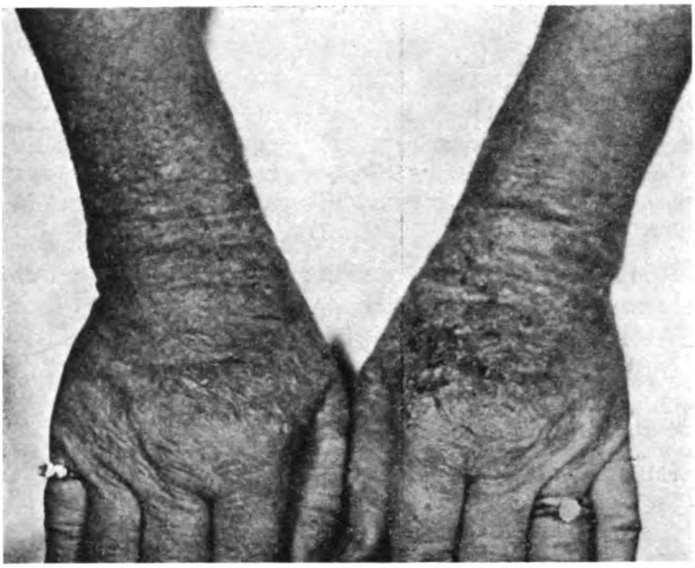

Fig. 80.—Contact dermatitis due to rubber gloves. At times the very measures used to protect an employee may be the cause of a dermatitis.

As the weeping and exudation subside, which they generally do within from forty-eight to ninety-six hours after the proper use of continuous wet compresses, soothing ointments such as the following may be used:

| | |
|---|---|
| Liquor aluminum acetate | 15.0 |
| Lanolin, anhydrous | 25.0 |
| Lassar's paste, plain | 20.0 |

Sig.: Apply locally two or three times daily. If some evidence of infection still persists, either ammoniated mercury (2 to 3 per cent) or sulfur precipitate (2 to 3 per cent) may be added to the above ointment.

When a stage of chronicity has been reached and the skin lesions are dry, scaly, and thickened, the use of some tar-containing preparation is indicated. In milder cases this may consist of ichthyol, 2 per cent in Lassar's paste. Later, a 5 per cent oil of cade ointment may be applied. The most useful of all the tars, however, is white crude coal tar paste (see Infections Eczematoid Dermatitis).

CONSTITUTIONAL THERAPY.—This is rarely of aid in occupational dermatitis. Dainow[28] has reported desensitization from the administration of vitamins A and D. In very stubborn cases, removal of foci of infection may be necessary.

**Infectious Eczematoid Dermatitis.**—Notoriously resistant to therapy, this moist, vesicular, eczematous condition may often be ameliorated or cured by the application of a 2 per cent aqueous solution of gentian violet twice daily. As long as the oozing persists, continuous wet compresses of from 0.25 to 0.5 per cent aqueous solution of silver nitrate may be used. Another highly satisfactory wet compress to use in this disorder is the following:

| Alibour water: | Gm. or cc. |
|---|---|
| Copper sulfate | 1.6 |
| Zinc sulfate | 5.6 |
| Saturated camphor water | q. s. ad. 240.0 |

Sig.: Use 2 tablespoonfuls of the above in a glass of tap water and use as a continuous wet dressing. Change dressing every two to three hours.

In addition, small doses of unfiltered roentgen rays are of value. In persons tolerant to tar, White's crude coal tar paste is often very efficacious, especially after the weeping has subsided:

| White's crude coal tar paste: | Gm. or cc. |
|---|---|
| Crude coal tar | 4.0 |
| Zinc oxide | 2.0 |
| Corn starch | 15.0 |
| Petrolatum | 15.0 |

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## CHAPTER XXXV

# OXYGEN THERAPY IN OCCUPATIONAL MEDICINE

Although the physical and chemical characteristics of the various gases have been studied for many decades, their therapeutic application did not occur until after the investigations of Van Slyke, Haldane, Boothby, Barcroft, Henderson, Haggard, Barach, and a few others. Barach,[1] in particular, has demonstrated the value of oxygen, carbon dioxide, and sometimes helium, in the treatment of cardiac failure, coronary artery disease, pneumonia, pulmonary edema, emphysema, atelectasis, and asthma. While general medicine has accepted this type of therapy, industrial medicine has not utilized it to the degree warranted.

Following an undue exposure to certain industrial chemicals, the pathology found in the lungs is not dissimilar from that in various respiratory and cardiac diseases of nonoccupational origin. In these latter pathologies the oxygen want is due to the inability of the lungs to diffuse oxygen, or of the heart to circulate oxygen in the blood. This is likewise true of the industrial agents which may act as asphyxiants, or as irritants to the lungs, or as anesthetics to the respiratory center in the brain. Regardless of the manner of action, the result is anoxemia. It is to combat this anoxemia that oxygen therapy is of value. When resorted to, oxygen-enriched atmospheres containing 40 to 60 per cent oxygen will raise the oxygen saturation of arterial blood to or near normal levels.

Carbon dioxide is likewise a common therapeutic agent. Ever since Haldane, Priestley, and Douglas contended that the volume of breathing is controlled by the percentages of carbon dioxide in the air which reaches the lungs, this gas has been employed to stimulate respirations. Yet, if the writer interprets the observations of Barach[1] correctly, the general use of carbon dioxide is not only of questionable value in many instances, but even likely to be harmful. Barach and co-workers would discourage its use in the treatment of most cardiac and respiratory diseases except for postoperative atelectasis, hiccoughs, and accidental asphyxia.

In Barach's recent excellent book he gives but scant space to the treatment of the occupational disturbances of the lungs. Yet it has been the writer's experience that oxygen therapy is of great value in immediate treatment of the acute effects from undue exposure to sulfur dioxide, phenol, ammonia, nitric acid, nitrous fumes, and cadmium. All of these are capable of producing a marked pulmonary edema. Carlisle[2] likewise found that oxygen gave striking results in the treatment of pulmonary edema due to chlorine and nitric acid.

Broadly, the chemical agents used in industry which may affect the respiratory system are:

1. The Irritants: The most common of these are chlorine, bromine, ammonia, nitrous fumes, chloropicrin, phosgene, diphosgene, sulfur dioxide, and cadmium. Exposure to high concentrations of these may result in pulmonary edema, bronchospasm, bronchopneumonia, and concentration of the blood in the pulmonary vessels.

2. The Asphyxiants: These are primarily carbon monoxide, cyanide, and hydrogen sulfide. In carbon monoxide, asphyxia is produced through the displacement of oxygen within the blood by the noxious gases. Therefore, the more quickly the disassociation is attacked and the normal relationship of oxygen to hemoglobin re-established, the less permanent will the damage be. It is in this type of case that carbon dioxide should be combined with oxygen in amounts from 5, 7, or 10 per cent. Carbon dioxide stimulates the respiratory center to produce full, deep respirations, thus relieving anoxemia.

Recently the investigations of End and Long[3] indicate that pure oxygen under pressure is of greater value than is the carbon dioxide-oxygen mixture. By animal experimentation they showed that carbon monoxide causes tissue damage by producing anoxia and that current methods of treatment are unable to correct this condition. Inhalation of oxygen under three atmospheres of pressure is capable of preventing such anoxia by causing solution of enough oxygen in the blood to provide for the needs of the tissues. Inhalation of oxygen under three atmospheres of pressure is safe for several hours.

The first action of the hydrogen sulfide is upon the respiratory center. In a person overcome by these fumes, respirations will cease before the heart action stops. It is, therefore, imperative to stimulate the respiratory center by carbon dioxide-oxygen inhalations and/or by artificial respirations.

Oxygen is of little or no value in cyanide poisoning, since cyanide, instead of combining with the hemoglobin in the blood stream, retards oxidation in the tissue cells.

3. The Anesthetics: A large group of solvents used in industry give off vapors which affect the central nervous system by producing anesthesia. Of these, the most common are carbon tetrachloride, trichlorethylene, and the petroleum distillates. In the average case these do not cause any pathology within the lungs proper, although the petroleum distillates have been known to cause pulmonary edema. The author saw a case in which pleural effusion resulted after a massive dosage of gasoline. Just as in operative anesthesia, an overdosage of these gases produces unconsciousness which demands immediate oxygen therapy.

It should be pointed out to the reader that in cases of pulmonary edema from any cause, occupational or nonoccupational, the mere utilization of oxygen is not sufficient. The best results are obtained only if oxygen is administered under positive pressure. While all physicians are acquainted with nasal catheter, various masks, oxygen tents, and oxygen rooms, the apparatus for positive pressure needs explanation:

"The Oxygen-injector Mask Metered for Positive Pressure is designed solely for the treatment of pulmonary edema due to cardiac-respiratory illness or gas poisoning. It should be employed cautiously in patients with shock.

"The injector mask has been equipped with a metal disk that surrounds the respiratory flutter valve and contains on its surface five orifices for varying diameters. Expiration proceeds naturally without pressure when the largest orifice is employed. When the disk is turned to progressively smaller orifices, the patient exhales under a positive pressure which is 1, 2, 3, or 4 cm. of water, depending upon the size of the orifice. These pressures have been recorded during quiet breathing, and would be higher in dyspneic patients. It will be found that expiration at a pressure of 4 cm. of water is uncomfortable and can be used for short periods only. However, a swifter clearance of pulmonary edema, especially in chlorine and nitric acid gas poisoning, may be obtained when the higher pressures are used for short periods. In pulmonary edema taking place during the course of clinical illness, 3 cm. of water may be adequate to result in a disappearance of the signs of moisture in the lungs and a smaller pressure of 1 to 2 cm. of water may then be used for periods of one to three hours, as may be required. Return of signs of moisture in the lungs indicates re-application of positive-pressure breathing.

"In order to employ 100 per cent oxygen, a high liter flow such as 12 to 15 liters per minute is necessary to prevent collapse of the collecting bag. Under these circumstances it is desirable for the injector to be one-third full of water so that the oxygen may pick up some moisture as it passes through. Test of the relative humidity of 100 per cent oxygen passing over the water surface in the injector has shown a relative humidity of 20 per cent of the air entering the mask. However, it has also been demonstrated that additional moisture is contributed to the inspired air because the inner surface of the mask is itself wet with moisture which has condensed on it from the expired air and the actual relative humidity of air entering the mask under these circumstances is 40 per cent. If a higher relative humidity is desired during the inhalation of 100 per cent oxygen, either with or without positive pressure, the injector may be removed and a water bottle attached to the regulator. The oxygen then passes through 2 or 3 inches of water and possesses a higher relative humidity.

"The patient may be treated with 50 or 60 per cent oxygen, and the positive pressure desired, by setting the injector and pressure dials at the appropriate orifices. This results in considerable saving of oxygen and is a therapeutically effective, efficient, and practical procedure. Although a swifter clearance of edema of the lungs may be obtained by expiration at high pressures, it is frequently desirable to employ lower pressures, such as 2 or 3 cm. of water, even though a longer time is necessary for complete clearance of the signs of edema. This is in part because of lessened discomfort to the patient, but also because low pressures do not significantly retard the entrance of blood into the right heart. In patients with cardiac illness, such as congestive heart failure, the retardation of blood is comparable to tourniquetting the extremity and facilitates the recovery of the heart muscle by making it possible for the heart to work on a smaller volume of blood.

"However, in patients with peripheral circulatory failure, pressure of 1 cm. of water may be employed in the attempt to eliminate pulmonary edema without causing any significant decrease in return of blood from the right heart. The systolic blood pressure should be carefully recorded at ten-minute intervals in all patients with shock. When pulmonary edema has cleared, the patient may breathe 60 to 100 per cent oxygen without pressure. If no recurrence of edema takes place and the patient is clinically better, the injector may be turned to 50 per cent oxygen and then to 40 per cent and the mask may be finally removed when the clinical condition of the patient indicates that he is out of danger. If pulmonary edema recurs after termination of positive pressure, the pressure should be applied again at 3 cm. of water and then gradually lowered at longer intervals."[1]

It is hoped that this rather concise discussion of oxygen therapy will focus the attention of the industrial physician upon an exceedingly valuable and often life-saving aid in the treatment of respiratory affections due to the inhalation of certain noxious vapors or fumes. Too frequently workers who are overcome by such agents are given ineffectual smelling salts, hypodermics of indiscriminate medication, or are subjected to an interim of neglect while awaiting transportation to a hospital. Nor is hospitalization always assurance that oxygen therapy will be instituted upon the patient's arrival, since the pathology of the occupational respiratory affections is not too well known by the attendant staff of the average nonindustrial hospital. The industrial physician would do well to specify the type of treatment he desires when referring cases to such institutions.

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# CHAPTER XXXVI

# SYNTHETICS

## Synthetic Rubber

In the desire to create substitutes for many of nature's materials, it has been possible to duplicate the natural product by laboratory synthesis. This is the fact with regard to practically all of the synthetic products except rubber. The sudden demand for rubber at the beginning of World War II necessitated the building of rubber plants all over the United States. To the average citizen it looked like a great magical stunt. With few exceptions, the workmen from these rubber plants were cared for by the neighborhood physician who was not even informed what chemicals were used in the production of synthetic rubber. These plants were often referred to as "butadiene" plants, but other chemicals were never mentioned, although a great number of them are required in the process. The raw materials used as a starting point for synthetic rubber, together with polymer building units and trade names for such, are given in Table XX.

The word Buna is derived from the first syllable of butadiene and the chemical symbol for sodium, Na, which is involved in the reaction. The S and the N (Buna S and Buna N) designations stand for sulfur and nitrogen, respectively.

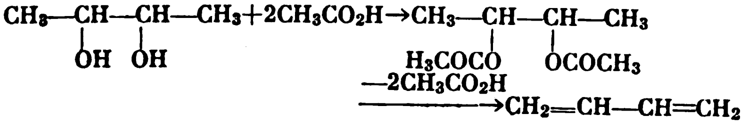
Of the various synthetic rubbers, the most common types are known as Neoprene, the Thiokols, Buna S, Buna N, and butyl rubber. A description of the essential raw materials is as follows:

Butadiene is obtained chiefly from petroleum gases. By a special cracking process, petroleum can be modified to give appreciable yields of butadiene largely by increasing temperatures and lowering pressures. Butadiene can also be obtained by catalytic hydrogenation of butane or butene.

Several processes are available[1] for the production of butadiene from ethyl alcohol or farm products. The Lebedev (Russian) method consists of catalytically converting two moles of ethyl alcohol to one mole of butadiene.

$$2C_2H_5OH \xrightarrow{\text{Catalyst}} CH_2{=}CH{-}CH{=}CH_2+2H_2O$$

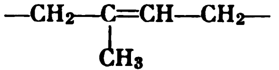
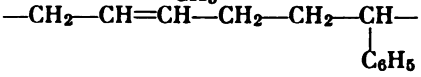
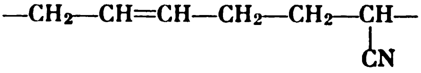
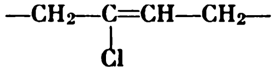
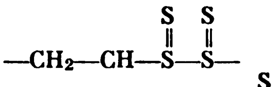
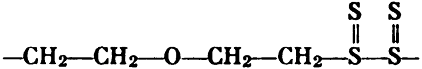
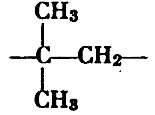
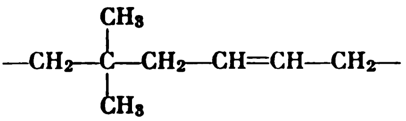
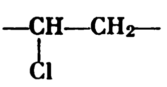
A new method consists of fermenting grain to 2,3-butylene glycol. Acetylation and subsequent deacetylation give good yields of butadiene.



$$CH_3{-}\underset{\substack{|\\OH}}{CH}{-}\underset{\substack{|\\OH}}{CH}{-}CH_3+2CH_3CO_2H\rightarrow CH_3{-}\underset{\substack{|\\H_3COCO}}{CH}{-}\underset{\substack{|\\OCOCH_3}}{CH}{-}CH_3 \xrightarrow{-2CH_3CO_2H} CH_2{=}CH{-}CH{=}CH_2$$

TABLE XX. CLASSIFICATION OF RUBBER AND RUBBERLIKE PRODUCTS

(Shreve: The Chemical Process Industries, New York, 1945, McGraw-Hill Book Co., Inc.)

| RAW MATERIALS | POLYMER UNIT | TRADE NAMES |
|---|---|---|
| **Latex from Hevea tree** |  $-CH_2-\underset{\substack{\vert\\CH_3}}{C}=CH-CH_2-$ | Rubber |
| **Butadiene + styrene** |  $-CH_2-CH=CH-CH_2-CH_2-\underset{\substack{\vert\\C_6H_5}}{CH}-$ | Buna S<br>GR-S rubber *(U.S. Gov.)*<br>Chemigum S *(Goodyear)* |
| **Butadiene + acrylonitrile** |  $-CH_2-CH=CH-CH_2-CH_2-\underset{\substack{\vert\\CN}}{CH}-$ | Buna N<br>Perbunan *(Stanco Products)*<br>Ameripol *(Goodrich and Phillips)*<br>Hycar *(Goodrich and Phillips)*<br>Chemigum N *(Goodyear)* |
| Chloroprene |  $-CH_2-\underset{\substack{\vert\\Cl}}{C}=CH-CH_2-$ | Neoprene *(Du Pont)*<br>Sovprene *(Russia)*<br>Mustone *(Japan)*<br>GR-M *(U.S. Gov.)* |
| **Ethylene dichloride + sodium tetrasulfide** |  $-CH_2-CH-\overset{\substack{S\\\Vert}}{S}-\overset{\substack{S\\\Vert}}{S}-$ | Thiokol A *(Dow)*<br>GR-P *(U.S. Gov.)* |
| **Dichloro-ethyl ether + sodium tetrasulfide** |  $-CH_2-CH_2-O-CH_2-CH_2-\overset{\substack{S\\\Vert}}{S}-\overset{\substack{S\\\Vert}}{S}-$ | Thiokol B *(Dow)*<br>Perduren *(Germany)* |
| **Isobutylene + (isoprene)** |  $-\overset{\substack{CH_3\\\vert}}{\underset{\substack{\vert\\CH_3}}{C}}-CH_2-$ | Vistanex *(Stand. Oil of N.J.)*<br>Oppanol *(I.G. Farbenindustrie)* |
| **Isobutylene + butadiene** |  $-CH_2-\overset{\substack{CH_3\\\vert}}{\underset{\substack{\vert\\CH_3}}{C}}-CH_2-CH=CH-CH_2-$ | Butyl *(Stand. Oil of N.J.)*<br>Flexon *(Stand. Oil of N.J.)*<br>GR-I *(U.S. Gov.)* |
| **Vinyl chloride + plasticizer** |  $-\underset{\substack{\vert\\Cl}}{CH}-CH_2-$ | Koroseal *(Goodrich)*<br>Korgel *(Goodrich)*<br>Flamenol *(General Electric)* |

The process employed in the 80,000 ton butadiene plant operated by the Carbide and Carbon Chemicals Corporation, at Institute, W. Va., makes the butadiene from ethyl alcohol. The alcohol is catalytically oxidized to acetaldehyde which is reacted hot with more alcohol in the presence of a catalyst to form the butadiene. The reactions may be presented as follows:

$$\underset{\text{Alcohol}}{C_2H_5OH+\text{air}} \xrightarrow{\text{Catalyst}} \underset{\text{Acetaldehyde}}{CH_3CHO+H_2O}$$

$$\underset{\text{Acetaldehyde alcohol}}{CH_3CHO+C_2H_5OH} \xrightarrow{\text{Catalyst}} \underset{\text{Butadiene}}{CH_2{=}CH{-}CH{=}CH_2+2H_2O}$$

It is probable that the acetaldehyde and alcohol combine to form 1,3-butylene glycol which loses water to give the butadiene. The reaction product is cooled in heat exchangers and condensers to furnish a crude butadiene, which is rectified by distillation from the uncracked raw materials, resulting in a 98.5 per cent pure butadiene.

*Chloroprene*, 2-chloro-1,3-butadiene, is made by treating vinylacetylene with cold, aqueous hydrochloric acid. The essential steps starting with acetylene are:

$$2HC{\equiv}CH \xrightarrow{\text{Catalyst}} \underset{\text{Vinyl acetylene}}{CH_2{=}CH{-}C{\equiv}CH} \xrightarrow[Cu_2Cl_2+NH_4Cl]{+HCl} \underset{\text{Chloroprene}}{CH_2{=}CH{-}CCl{=}CH_2}$$

*Styrene*, vinylbenzene, is prepared starting from benzene and ethylene by a Friedel-Crafts reaction. This is dehydrogenated by mixing with steam at 800° C. in the presence of a bauxite catalyst. The first product is ethyl-benzene which is catalytically dehydrogenated to yield styrene.

*Acrylonitrile* is made by the dehydration of ethylene cyanohydrin, which may be prepared by adding hydrogen cyanide to ethylene oxide or by treating ethylene chlorohydrin with sodium cyanide.

*Neoprene*. The reactions by which this first one of the synthetic rubbers was successfully made are as follows:

$$\underset{\text{Acetylene}}{2CH{\equiv}CH} \xrightarrow{\text{Catalyst}} \underset{\text{Vinyl acetylene}}{CH{\equiv}C{-}CH{=}CH_2} \xrightarrow{HCl} \underset{\text{Chloroprene}}{CH_2{=}CCl{-}CH{=}CH_2}$$

$$\underset{\text{Chloroprene}}{nCH_2{=}CCl{-}CH{=}CH_2} \text{ polymerizes to } \underset{\text{Neoprene}}{({-}CH_2{-}CCl{=}CH{-}CH_2{-})\,n}$$

These reactions require exact control to carry out successfully on a manufacturing scale, as compounds are being handled that can decompose with violence. Certain amounts of acetylene and of divinylacetylene are distilled in the course of the making of the vinylacetylene.

*Butyl rubber.* This has excellent properties of chemical inertness and low permeability to gases that make it desirable in special applications. It is copolymerized from isobutylene and isoprene.

*Thiokol.* The condensation of ethylene dichloride and sodium tetrasulfide and the subsequent polymerization are somewhat different. A sodium tetrasulfide solution is added slowly to the ethylene dichloride. Magnesium hydroxide is introduced into the polysulfide solution as a dispersing agent. The temperature is held at 80° C., and the mixture is agitated vigorously for five hours. This produces a latex in which the solid material gradually settles out. The excess water is decanted, the solid material is washed several times with water, and finally hydrochloric acid is added to coagulate the rubber. The rubber forms in a thick sheet at the bottom of the vessel. After removal of the solution, workmen cut out the rubber from the vessel. The product is then dried and shredded and is ready for mastication.

Faced with the problem of producing synthetic rubber for general uses and not for specialty articles, the United States will doubless improve the present synthetics and their compounding in the next few years, particularly since many people are employed in research on this subject. It is too early to evaluate these synthetic resilient products as to both competitive cost and competitive properties, although in World War II synthetic rubbers have been of fundamental importance and have relieved a national emergency.

Buna S type resembles natural rubber most closely and is employed chiefly for the fabrication of tires that approach those made from natural rubber in their road wear.

Other synthetics have specific properties that make them superior in certain fields. The first commercial example of this is Neoprene, the oil resistance of which in prewar years enabled it to sell at several times the price of natural rubber for hose to transport oil and gasoline and for gasket material. Neoprene also possesses superior resistance to flame or degrading influences of sunlight and air, caused probably by the chlorine content of the Neoprene.

On the other hand, butyl rubber (as well as other isobutylene products) is quite soluble in petroleum hydrocarbons but, because of its almost complete lack of unsaturation, it is chemically very resistant to attack by acid, alkalies, and ozone. Its vulcanizates are highly impermeable to gases and are being proposed for inner tubes.

Another example of a specific application is the use of Thiokol FA to line concrete tanks employed for gasoline and oil storage. The Thiokol is not attacked by the petroleum products and thus prevents loss, and also it protects aviation gasoline from the drop in octane rating that the alkali of the concrete would bring about.

Butadiene vapors are strongly irritant to the skin, eyes, and upper respiratory passages. It has been the experience of this author that at the Los Angeles rubber plant during World War II, most of the contacts with butadiene were insignificant. The effect upon the skin was usually delayed, which means that the burning sensation was not immediately apparent to the workman. Whenever the workman became cognizant of having been in contact with this substance, he was immediately placed under a shower of water, and his eyes were irrigated copiously with water. No systemic reactions were ever encountered.

Styrene (vinylbenzene) is chemically similar to toluene and is presumed to have the same toxic effects, although to the author's knowledge no incidence of intoxication from this substance has occurred in the rubber industry. It is often irritating to the skin and respiratory tract.

Neoprene (Chloroprene) is readily absorbed through the skin. It has narcotic properties and is especially injurious to the liver and, to a lesser extent, to the kidneys.

Vinyl chloride (chloroethylene) may produce dizziness, headache, and mild narcotic effects. It is considered less toxic than either chloroform or carbon tetrachloride.

Carpenter and associates[2] conducted studies on the inhalation of 1,3-butadiene and compared the narcotic effects with benzol, toluol, and styrene. Their summary and conclusions were:

"1. Butadiene in concentrations of 600, 2300, and 6700 p.p.m. caused no significant progressive injury to small animals during an 8-month exposure period, 6 days a week, 7½ hours per day, although the highest concentration retarded growth slightly and caused light cloudy swelling in some livers. Data on fertility, blood cytology, body weight and length, organ weight, blood chemistry, urinalyses, and micropathology are discussed in support of this statement.

"2. The iodine pentoxide method of analysis has been adapted to the determination of butadiene in air and in blood.

"3. The distribution coefficient for butadiene between blood and air was found to be 0.603 *in vitro* and 0.645 *in vivo*. This substantial agreement indicates that the passage of butadiene from inspired air into the blood is a process of simple diffusion of the gas from the alveoli and simple solution in blood.

"4. Anesthetization experiments on rabbits were carried out to compare the symptoms, reflexes, and blood levels of butadiene, benzol, and toluol. The narcotic action of butadiene was less than that of the other two.

"5. Investigation of the urine of rabbits after chronic and acute exposure failed to reveal the presence of any metabolic products of butadiene. The increase in urinary hippuric acid excretion in humans following exposure to toluol was verified, and suggestive information obtained along the same line following styrene inhalation was noted.

"6. Two human subjects exposed for 4- to 8-hour periods agree that 800 p.p.m. styrene is the most objectionable vapor they used, 800, 600, 400, and 200 p.p.m. toluol are next in that order, and 8000 p.p.m. butadiene had an effect no greater than 200 p.p.m. of toluol. On the basis of psycho-motor effect, which may indicate a tendency to carelessness and accidents, 8000 p.p.m. butadiene is no worse than 200 p.p.m. toluol.

"7. Butadiene is practically innocuous aside from its narcotic effect at very high concentrations and has little if any cumulative action. Provided the lower explosive limit is not exceeded, it is unlikely that the health of anyone inhaling its vapors will be impaired, although many may find its odor objectionable. Humans accidentally anesthetized by extremely high concentrations should return quickly to normal if they are rescued while respiration and heart action are still strong. Deep anesthesia, however, is likely to terminate fatally in a short time."

In addition to the aforementioned raw materials used in the synthetic rubber industry, the workman must be protected against undue exposure to a number of involved solvents. These solvents are petroleum naphtha, benzol, toluol, xylol, trichlorethylene, carbon tetrachloride, ethylene dichloride, propylene dichloride, and methyl ethyl ketone. The inherent toxic properties of each one of these has been discussed in the chapter on Solvents.

There are also a number of compounds, organic accelerators, and antioxidants which deserve brief mention. As yet little is known about certain of these with regard to their toxic effects.

*Acetaldehyde Aniline.*—

*Composition:* A condensation product of acetaldehyde and aniline.

*Use:* General accelerator.

*Health hazard:* No definite report on the toxicity of this material is available. There have been no reports of trouble in handling it. However, it should be tested for free aniline which would introduce a serious hazard if not completely removed.

*Albasan.*—

*Composition:* A mixture of organic bases and b-naphthol.

*Health hazard:* b-Naphthol is a local irritant to the skin and mucous membrane and may be absorbed through the skin in sufficient quantities to cause irritation of the kidneys and internal injuries to the eyes. Contact with the skin should be avoided and the dust should be controlled by ventilation.

*A-Naphthylamine.*—

*Composition:* A pure compound occurring as a white crystalline powder with a specific gravity of 1.223 and melting point of 50° C. It rapidly oxidizes and turns dark upon exposure to light and air. The odor is characteristic and unpleasant.

*Use:* Comparatively weak accelerator, not widely used because of its disagreeable odor.

*Health hazard:* There is some evidence that long-continued absorption of small amounts of naphthylamine has produced bladder tumors in dye workers. Although this is still questionable, there is no doubt that it is considerably less active than the b-naphthylamine. In larger concentrations it produces irritation of the skin and mucous membranes and also the blood and central nervous system, effects characteristic of amino compounds.

*Aldehyde Ammonia.—*

*Composition:* An aldehyde-amine formed by the direct addition of ammonia to acetaldehyde. It is a white crystalline solid which rapidly turns to a brown resin when exposed to the air and readily decomposes into ammonia and acetaldehyde upon heating.

*Use:* Medium strong accelerator which has been largely replaced by more stable materials.

*Health hazard:* When heated during mixing and calendering operations, aldehyde ammonia decomposes and releases acetaldehyde and ammonia. The acetaldehyde is an irritant to the skin and mucous membranes and is extremely nauseating to some men, but they are said to become accustomed to it. No systemic toxic effects have been reported. Exhaust ventilation should be used at the mixing and calendering operations when acetaldehyde is in the compound. The odor is not released at other operations.

*b-Naphthylamine.—*

*Composition:* A pure compound occurring as a white crystalline powder with a specific gravity of 1.06 and melting at 112° C. It oxidizes rapidly upon exposure to light and air and turns brown.

*Use:* Accelerator, slightly stronger than aniline but no longer much used.

*Health hazard:* b-Naphthylamine is considerably more poisonous than the alpha compound. Cases of blood destruction and kidney and bladder injury have been reported in the chemical industry. It has also been indicated as the cause of bladder tumors in dye workers. No cases of poisoning have been reported in the rubber industry.

*b-Naphthol.—*

*Composition:* A pure compound with a melting point of 121.6° C. and a boiling point of 285° C. It is a white powder when pure but oxidizes and darkens on exposure to light and air.

*Health hazard:* b-Naphthol is moderately toxic although somewhat less than a-naphthol. It is an irritant for the skin and mucous membranes, and a sufficient amount may be absorbed through the skin to cause irritation of the kidneys and internal injury to the eyes.

*Butyraldehyde-Aniline.*—

*Composition:* A reaction product of butyraldehyde and aniline, usually furnished as a tan powder.

*Use:* Antioxidant for uncured rubber, dry rubber, latex compositions, and Neoprene. An activator for rubber and Buna S.

*Health hazard:* This material is believed to be relatively safe when pure. One company reporting states that traces of aniline, a source of a serious hazard, have been found in some samples. Eighteen companies and two manufacturers of products of this type state that they have had no trouble with toxicity.

*M-Toluylene-Diamine.*—

*Use:* To a small extent in rubber and in the formulation of antioxidants.

*Health hazard:* Toluylene-diamine appears to have three different types of action upon the body. It acts as a poison to the central nervous system, produces jaundice by action on the liver and spleen, and produces anemia by destruction of the red blood cells. This is quite similar to the action of aniline, although not identical with it.

The high boiling point and the fact that the material is a solid make it somewhat less hazardous than aniline at room temperature, although it can be absorbed by inhalation at higher temperatures. Von Oettingen refers to a fatal accident in which a workman was exposed to vapors and possibly to skin contact with the compound. He suffered from nausea and vomiting, showed jaundice, and developed stomach disturbances. Von Oettingen also refers to a case of permanent injury to the eye from the use of toluylene-diamine as a dye for the eyelashes.

*Methylene Aniline.*—

*Composition:* Methylene aniline, also called formaniline, formaldehyde aniline, and anhydro formaldehyde aniline, is prepared by the action of formaldehyde on aniline. Two products are possible, depending upon whether one or two molecules of aniline enter into the reaction. If two molecules of aniline per molecule of formaldehyde react, the product is methylene diphenyldiamine. Methylene aniline is the product when one molecule of aniline reacts with one molecule of formaldehyde. It is a white powder melting at 133° to 137° C. and is fairly stable, although it turns brown by oxidization when exposed to air and light.

*Use:* Mild accelerator, only slightly more powerful than aniline and requiring about 2 per cent on the rubber. It also acts as a softener and a secondary accelerator, particularly with the guanidine accelerators.

*Health hazard:* Commercial methylene aniline often contains traces of uncombined aniline or formaldehyde and some methylene diphenyldiamine. Since these materials are absorbed through the skin to produce systemic poisoning and will also produce a severe dermatitis, the material should not be permitted to come in contact with the bare hands. Some volatilization takes place during mixing and calendering, producing a pungent odor and irritation of the eyes and nose and throat. Mills and calenders should be hooded to remove vapors.

*p-Phenylenediamine.*—

*Composition:* A pure compound occurring as a white crystalline powder melting at 140° C. It very readily oxidizes and turns grayish-brown on exposure to air.

*Use:* p-Phenylenediamine has been very extensively used as a dye for hair and fur. It is a powerful accelerator for rubber.

*Health hazard:* This is a common cause of skin irritation and to a lesser extent, of bronchial asthma, In addition to the tendency to produce dermatitis and asthma, p-phenylenediamine is decidedly poisonous and should be handled with extreme care. The operators weighing it out should wear rubber gloves, and either local exhaust ventilation or respirators should be provided to prevent breathing the dust.

Mills and calenders should be hooded and exhausted, since the p-phenylenediamine fumes during mixing and calendering, and these fumes are injurious to both the eyes and respiratory organs.

*Tetramethyl Thiuramdisulfide.*—

*Composition:* It is a nonhygroscopic, crystalline substance with a melting point of 150° C. It is prepared by oxidizing the reaction product of dimethylamine and carbon disulfide.

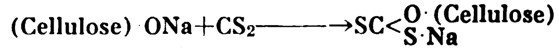
*Use:* It is a powerful accelerator for rubber and Buna S and a retarder for Neoprene. Zinc oxide is usually used as an activator.

*Health hazard:* Of 25 users, eight report some trouble with this compound. Dermatitis occurs in some instances, in addition to irritation of the eyes, nose and throat. Precautions advised include prevention of contact with the dust and the use of gloves and long-sleeved undershirts. On some jobs a filter respirator may be required to prevent nose and throat irritation.

## Synthetic Fibers

Nitrocellulose was the first of the synthetic fibers, but since its introduction there have been added viscose rayon, acetate rayon, cuprammonium rayon, Aralac, Fiberglas, Teca Nylon, and Vinyon.

**Rayon** filament yarn is made by three methods, the viscose process, the cellulose acetate process, and the cuprammonium process. The viscose process is the one most widely used. The principal raw materials[3] used by this process are dissolved wood pulp, refined cotton linters, carbon disulfide, sulfuric acid, caustic soda, glucose, ammonia, magnesium sulfate, sodium carbonate, sodium aluminum sulfate, sodium sulfide, zinc sulfate, and other chemicals in smaller quantities. This process is based on the discovery made in 1892 by two English chemists, Gross and Bevan, that mercerized, or alkali, cellulose forms a definite, though unstable, compound with carbon disulfide. This compound, called cellulose xanthate, is a yellow solid and its formula may be written:



$$\text{(Cellulose) ONa}+\text{CS}_2 \longrightarrow \text{SC}<\begin{matrix}\text{O}\cdot\text{(Cellulose)}\\ \text{S}\cdot\text{Na}\end{matrix}$$

Cellulose xanthate

Cellulose xanthate is soluble in dilute caustic soda, and from this solution the regenerated cellulose is precipitated by the action of an acid. Simply stated, the chemical reactions of this process are as follows: Alkali cellulose is combined with carbon disulfide, and the resultant is dissolved in dilute caustic soda, producing a viscous solution called viscose. This solution is then sent to the spinning device and the emerging fine streams of the solution are coagulated by a bath containing sulfuric acid and other chemicals.

The sources of cellulose for this process are either wood pulp or refined cotton linters pulp. The American viscose rayon industry at present depends more on wood pulp than on cotton linters pulp for its cellulose supply. The alpha-cellulose content of wood pulp for this process ranges from 88 to 92 per cent. Especially refined cotton linters pulp approximates 98 per cent alpha-cellulose content. Wood pulp and cotton linters pulp arrive at the rayon-producing plant in bales of white sheets. These sheets of pulp are stored in pulp storage rooms and held for several weeks in an atmosphere of controlled temperature and humidity.

When ready to be put into process, the pulp is carefully selected for steeping, which is merely a mercerization process. The pulp is placed in a steeping press which is then filled with a NaOH solution of 17 to 18 per cent concentration at a definite temperature. The pulp is allowed to soak for about two hours. In this stage of the process the $\alpha$-cellulose remains insoluble, but the $\beta$- and $\gamma$-cellulose are leached out by solution with the caustic soda. The sheets of cellulose are then pressed together by a hydraulic ram to squeeze out the caustic soda, which in turn is drained out of the tank. The resulting product is called alkali cellulose. The liquid drained out of the storage tank is sent to dialyzers for purification and re-use.

The soft alkali cellulose sheets are then dropped into shredder machines where the alkali cellulose is reduced to soft crumbs by a shredding operation, which loosens, blends, and fluffs up the cellulose fibers. During this entire operation the alkali ecllulose is kept under a temperature of about 18 to 20° C. The crumbs are then removed from the shredder and put into steel cans. These containers are sent to a temperature-controlled aging room where the cellulose undergoes a change in its molecular structure during the several days of aging.

After aging, the alkali cellulose is ready for xanthation. In this operation the alkali cellulose is converted into another chemical compound, cellulose xanthate, by treating it with carbon disulfide for several hours under controlled temperatures. This operation is performed in a churn which rotates horizontally as the carbon disulfide is introduced. As it is absorbed by the cellulose the color of the cellulose changes from white through gradations of yellow to orange. When the latter color stage has been reached, the xanthating process is completed. The excess carbon disulfide vapors are drawn off and the xanthate discharged into a viscose mixer or dissolving machine. This dissolving machine is filled with dilute caustic soda before receiving the charge of xanthate. The xanthate is dissolved in the NaOH solution and the resulting solution contains about 7.25 per cent cellulose and 6.5 per cent sodium hydroxide. It is brownish-green in color and viscous in character; hence the name viscose.

If no delustering agents are added, this solution will be used for spinning bright viscose yarn. For dull and semi-dull yarns there is added at this stage a dispersion of titanium dioxide whose average particle size by surface mean is 0.56 micron. The delusterant becomes an integral part of the yarn and, therefore, is permanent; it can never be rubbed out or washed off. Delustering is possible because titanium dioxide has a greater refractive index than that of the regenerated cellulose.

**Nylon.**—Nylon yarn is a new synthetic fiber which comes closer to silk in constitution and properties than any other synthetic fiber previously manufactured. Nylon is already well known to the general public because it has gone into a considerable volume of nylon hose and other articles of wearing apparel.

Nylon is a generic name applied to all superpolymers made from long-chain amino acids or from long-chain dibasic acids combined with diamines. Nylon 66 is the specific nylon now made on a large scale; it is the superpolymer formed by the combination of adipic acid and hexamethylene diamine. These two substances, both solids at room temperature, are mixed in solution to form nylon salt. Stabilizing agents are added to control molecular weight and viscosity; the solution is evaporated and the liquid residue transferred to an autoclave in which it is heated for many hours. The product is a melt, consisting of the final superpolymer. The melt is extruded in ribbon form onto a chilled wheel (casting wheel); the congealed ribbon, now a tough, horny material, is cut into small chips convenient for storage. To make the fiber, the chips are melted by passing them between heated grids; the liquid is fed to a pump and is supplied under the pressure of the pump to spinnerets, which form the filaments. The latter are congealed at once by a gentle blast of air and wound on bobbins. Next, the primary filaments, rather useless in their existing form, are cold-drawn to about four times their original lengths, with a decrease in diameter. By this operation, their properties are changed completely, for now they form elastic fibers, transparent, lustrous, and of high tensile strength. The explanation for the change is that the original random arrangement of the superpolymer molecules gives place to an oriented one. The cold-drawn filaments are twisted, sized, and rewound on spools or cones, ready for the textile mill.

Nylon fiber may be made into any of the standard deniers. The diameter of the individual filaments is governed by controlling the rate at which the molten nylon is pumped to the spinneret, and by the rate at which the filaments are drawn away. The filaments from one spinneret are united to form the yarn; the number of filaments in the yarn is determined by the number of holes in the spinneret. Filaments of extreme fineness can be made from nylon, much finer than those of ordinary textile fibers.

Nylon may be delustered as rayon is, by the addition of finely powdered (colloidal) titanium dioxide or similar pigments. Nylon may be colored in the molten mass, or the yarn, or finished fabrics may be dyed, preferably with acetate rayon dyes.

Nylon textile monofils are comparatively thick single filaments and serve for making supersheer hosiery and insect screening and for other purposes. Nylon solutions permit the coating of metal wires for insulation. Nylon molding powder is being developed for injection-molding equipment. Nylon fiber is used for shroud lines and canopies for parachutes, and for other military purposes.

Nylon is not soluble in water, nor in most of the ordinary organic solvents; it dissolves in phenol, cresol, and formic acid. The filaments may be dry-spun with such a solution, as acetate rayon is, but because of the nature of the solvents this is not desirable.

The two raw materials for nylon 66 had to be manufactured by new processes, for until this development, adipic acid and hexamethylene diamine were not made commercially in this country. Adipic acid, $COOH{\cdot}CH_2{\cdot}CH_2{\cdot}CH_2{\cdot}CH_2{\cdot}COOH$, is made from phenol. First, the phenol is hydrogenated to cyclohexanol, with the aid of a contact catalyst, at elevated temperatures. Next the cyclohexanol is oxidized, also catalytically, to give adipic anhydride, which is readily hydrated to the acid. Hexamethylene diamine is made from adipic acid by treating it catalytically with ammonia, to form the dinitrile, and hydrogenating the latter to give the final diamine, $NH_2{\cdot}CH_2{\cdot}CH_2{\cdot}CH_2{\cdot}CH_2{\cdot}CH_2{\cdot}CH_2{\cdot}NH_2$, a solid at room temperature.

The linear polymer forms by the combination of the end group of the acid with the end group of the diamine, leaving at each end of the new molecules reactive groups which undergo similar combinations. The first compound would be $COOH\cdot(CH_2)_4\cdot CO\cdot NH\cdot(CH_2)_6\cdot NH_2$.

**Cellophane, Sealing Caps.**—Transparent paper (Cellophane, Transparit, Sylpharp) is made by extruding a viscose solution through a long narrow slit into a setting bath. Kodapak is a cellulose acetate sheet of high transparency. The size of the slit determines the thickness the paper will have; the length of the slit is also the width of the paper. The process is continuous, and a sheet of any length is produced. It passes through the setting bath, through water to remove acid, through a bicarbonate solution to neutralize the last traces of acid, through a soda ash solution to remove sulfur, through a dilute acid to remove the carbonate, through water, then through a glycerine solution from which is absorbed 17 per cent glycerine. It is then dried on steam drums and rolled, very much as paper is. The transparent paper may be colored by passing the paper in the gel state, that is, right after alkali and acid removal, through a dye solution. It may be embossed by passing it between engraved copper rolls.

Cellophane is made moisture-proof and water-proof by passing the sheet, after the glycerine bath, through a bath consisting of a dilute lacquer solution with rather volatile solvents such as ethyl acetate. The sheet in loop form continues to a chamber wherein warm air removes the solvents; the lacquer with the proper amount of plasticizer and softeners remains as a coating. The solvents are recovered by means of activated carbon.

An artificial sausage casing has been made successfully for some time and marketed; it is seamless and is made in the same way as transparent paper.

Hydrated cellulose and cellulose acetate may be formed into caps for sealing bottles. The caps are kept under water until needed. In order to apply a cap, it is placed loosely over the stoppered neck of the bottle and left over night. It shrinks and attaches itself firmly to the neck of the bottle, sealing it.

**Cellulose Sponge.**—A strong, durable sponge is made from viscose, by incorporating Glauber salt crystals of all sizes, placing boxes with the mixture in the coagulating bath, and precipitating the cellulose. Some hemp or other fibrous material may be mixed with the viscose. The blocks so formed are leached with warm water, removing the crystals, which then leave corresponding cavities. An improvement consists in coagulating with a hot salt solution, which penetrates quickly into the interior of the blocks and also causes the contained Glauber salt to melt and thus be simultaneously removed. The blocks are cut into square-edged pieces, about right for one's hand. They take up water and may be squeezed free from it just like a sponge. A cellulose sponge with uniformly fine cellules is made by using fine crystals exclusively.

## The Plastics

According to Shreve,[4] plastics and resins may be defined as organic materials that can be easily molded or shaped by mechanical or chemical action to give tough, noncrystalline substances that are solid at ordinary temperatures. These solids may have various substances incorporated with them to impart color and strength. They are resistant to the action of many chemicals and atmospheric conditions.

The use of a plastic material for any specific application is dependent upon its composition, its particular properties, and the design of the part. Synthetic resins are the largest source of plastics, with cellulose derivatives ranking next. The great utility of plastics may be shown by reference to a few typical applications in the various fields where these new materials are being applied. In the automotive and airplane industries, many different plastics find specific utilization because of beauty, strength, oil and electrical resistance. Their resiliency and strength make them very useful in the manufacture of safety glass, laminated gears, cams, pulleys, self-lubricating bearings, plywood, and the like. In other fields where the plastic cannot be used itself, it is employed in combination with metals, as in the manufacture of steering wheels and plastic-covered dashboards. These combinations develop the best properties of each material.

Plastics are made from molding compositions which are prepared from two or more of the following raw material groups:

*Binder.*—This is usually a resin or cellulose derivative.

*Filler.*—Cellulose, wood flour, cotton fiber, asbestos, mica, or fabrics may be added to increase strength.

*Plasticizer.*—Plasticizers are added to cellulose derivatives and to certain thermoplastic resins to produce tough, resilient products of varying properties. These plasticizers may be camphor, phthalates, glycol esters, glycol ester-ethers, phthalyl glycollates, or fire retardants such as triphenyl and tricresyl phosphates.

*Dyes and Pigments.*—The dyes used vary in resistance to sunlight and to the plastic binder. The colors employed are the triphenylmethane basic dyes, azo dyes, and anthraquinone vat dye.

*Catalyst.*—The thermosetting resins may use either an acid or basic catalyst, depending upon the desired properties, while the resins of the vinyl group may use peroxides to initiate the polymerization of the monomers.

*Lubricants.*—Lubricants, such as stearates and metallic soaps, are used particularly in cold-molding compounds to facilitate the molding operation.

## Thermosetting Resins

### Phenolic Resins.—

MOLDING TYPE.—The phenolic resins comprise the largest group of plastics and are of coal tar derivation. They may be made from almost any phenolic body and an aldehyde such as formaldehyde or furfural. The resinous product obtained from the condensation of phenol and formaldehyde depends primarily on the concentration of the reactants, the nature of the catalyst used, the time of reaction, and the temperature. The outstanding properties of these resins are excellent heat resistance, water resistance, resistance to chemicals, moldability, and electrical insulating qualities. They do, however, lack color stability. They are used for automotive and aircraft parts, electrical insulation, corrosion-resistant apparatus, and radio cabinets and parts, as well as for telephone equipment.

The molding type of phenolic resins are of the "two-step" variety. An acid catalyst is used for the reaction of a limited amount of formaldehyde with excess phenol. The resulting thermoplastic resin is later mixed with more aldehyde in the presence of a basic catalyst such as hexamethylenetetramine, known in this industry as *hexa.*

Recently (1943) the heatronic molding process has been announced by the Bakelite group as an improvement in the molding or curing of plastics. This is particularly applicable to the molding of articles with a thickness of over 3/8 inch which are pressed as usual, then heated to the necessary 320° F. by employing a high frequency current as the source of the heat. This new method of curing causes the reactions to take place in less time and with much more uniform results.

CAST TYPE.—This type of phenolic resin is made by the direct or one-step process. The properties vary somewhat from those of the molded phenolic variety. It is possible to prepare these in a wide range of color, transparency, or opacity, largely because the resinification reaction or the curing is not carried to the completion desired in the ordinary infusible and insoluble phenol-formaldehyde resins. In addition they are readily machinable, have high tensile and impact strengths, and are nonflammable. Some of the more important uses are for industrial cabinet housings, novelties, decorative articles, interior lighting fixtures, adhesives for wood veneers and grinding disks, kitchen utensils, and handles. The phenol and formaldehyde with a basic catalyst (usually barium hydroxide or caustic soda) are placed in a nickel or stainless-steel reaction kettle and heated to the boiling point as low as 160° F. for a period of ten minutes to three hours. During the reaction, water separates as the upper layer, and the mixture darkens slightly and increases in viscosity. The degree of reaction is controlled by means of time, temperature, pH readings, and viscosity determinations. During the removal of about 75 per cent of the water under vacuum, the resin becomes sirupy.

At the proper time, while the resin is still hydrophilic, an organic acid (lactic acid, phthalic anhydride, or maleic acid) is added to neutralize the resin and to clarify the color. If the resin is still hydrophilic, after neutralization the now four-component resin should not separate from the remaining water when cooled to 75° F. If a translucent resin is desired, glycerine is added to the four-component resin after the lactic acid addition on the basis of 130 lb. per 1,000 lb. of phenol. Before the final dehydration of the resin, plasticizers, pigments, and colors are added to the kettle and mixed with the resin. Dehydration is effected under vacuum at a resin temperature not in excess of 165 to 175° F., and the hot resin is withdrawn and poured into preheated lead molds. The final reaction and hardening take place by curing the resin in the molds at 185° F. for periods up to five days. The curing ovens are heated with steam under precise temperature control.

The molds used in the casting of these resins are either single molds or the split type and may be made of lead or rubber backed with plaster of Paris. The castings are so made that the finished product is obtained by one or more standard machining operations including slicing, sawing, cutting, drilling, threading, forming, carving, embossing, turning, milling, and polishing.

**Urea-Formaldehyde Resins.**—When urea is condensed with formaldehyde, water-soluble, water-white intermediates are formed, which have been identified as monomethylol urea, $NH_2 \cdot CO \cdot NHCH_2OH$, and dimethylol urea, $CH_2OHNH \cdot CO \cdot NHCH_2OH$. Urea and formaldehyde, with some ammonia to control pH, react under controlled time and temperature, 60 to 100° C. These intermediates are used in admixture with some form of cellulose such as paper, pulp, or rayon before the final reaction and curing to an infusible, insoluble product.

The final curing takes place in the presence of suitable catalysts and at a controlled temperature. In combination with finely divided $\alpha$-cellulose, these resins are available as molding powders or granules. This material is compression-molded at temperatures from about 285 to 315° F. and at pressures varying from 3,000 to 6,000 pounds per square inch. The rate of setting or polymerization depends on the temperature used, the curing agents present, and the form of the finished part. The outstanding properties are unlimited translucency or opacity, excellent light diffusion, rigidity, and lack of odor or taste. These resins are used for closures, lighting reflectors, tableware, buttons, and baking enamels.

**Melamine Resins.**—These are formed by reacting melamine with formaldehyde to yield water-white, water- or water-alcohol-soluble intermediates. Melamine is obtained from dicyandiamide.

Molded or laminated parts made from melamine resins are extremely hard and resistant to abrasion. Molding powders made from these resins are molded by compression. The rate of curing is dependent on the same variables that control the curing rate of the urea resins. The cured resins are colorless, resist hot water and organic solvents, and are heat-resistant.

**Alkyd Resins.**—The resins grouped in this class are chiefly the reaction products from polybasic acids and polyhydric alcohols. However, the name of this class includes also those resins from polyhydric alcohols and polybasic acids that have been modified with fatty acids from vegetable oils, with rosin, and with other synthetic resins.

The reaction of glycerine or ethylene glycol with a polybasic acid like phthalic anhydride produces resinous products; when ethylene glycol is employed, the product is of a balsamlike appearance and is nonhardening; when glycerine is reacted, the product is thermosetting. Because most of this type of resin is made from glycerine and phthalic anhydride, the class is placed among the thermosetting resins. This thermoreacting quality is probably caused by cross linkages set up by more points of reaction of the glycerine. The introduction of modifiers, such as monobasic acids or monohydric alcohols, reduces the number of active points of reaction that play a part in forming cross links between linear polymeric chains and increases the solubility of the resin.

These resins are used chiefly in protective coatings rather than in molded plastics because of their poor moldability. When they are combined with urea resins, coatings of very superior gloss, hardness, and increased resistance are obtained. Particularly useful properties of these resins are long retention of initial good appearance, long exterior-exposure life on weathering, ease of application, solubility in relatively cheap solvents, high gloss, adhesion to metal, and quick drying or baking schedule.

**Thermoplastics Based on Cellulose.**—Cellulose is composed of long chains of these units which may number 3,000 to 3,500. These elongated chains and the reactions of the cellulosic polyhydric alcohol are responsible for the formation of tough, flexible, cellulosic plastics. The properties of the cellulose derivatives depend upon the substituent groups, the amount of substitution, the type of pretreatment, and the degree of degradation of the long chains into shorter lengths.

**Cellulose Ester Plastics.**—Cellulose plastics are usually solid or colloidal solutions of esters in plasticizers. The esters are made by esterifying purified, pretreated cellulose with appropriate acids and acid anhydrides in the presence of catalysts under controlled temperature. Both inorganic and organic esters are available commercially. The most important are the nitrate, acetate, and a mixed acetate-butyrate.

Fully *nitrated cellulose* is unsuited for a plastic base because of its explosive character. Hence there is made a partly nitrated product with a nitrogen content of about 10.5 to 11.5 per cent. This cellulose nitrate is placed in large kneading mixers with solvents and plasticizers and thoroughly mixed. The standard plasticizer is camphor. The compounded mixture is then strained under hydraulic pressure and mixed on rolls with coloring agents. The material is pressed into blocks. Finally, the plastic is made into sheets or other shapes seasoned to remove the residual solvent and polished by pressing under low heat. Some of the outstanding properties of cellulose nitrate include toughness, water resistance, ease of fabrication, ease of cementing, clarity, colorability, and inflammability. It is used for fountain pens, golf club parts, toys, novelties, toothbrush handles, and cutlery handles.

**Cellulose Acetate.**—Fully acylated celluolse is partly hydrolyzed to give an acetone-soluble product, which is usually between the di- and tri-ester. This hydrolysis takes place in aqueous organic acids in the presence of a catalyst under controlled time and temperature. These cellulose organic esters are precipitated in a large volume of water, washed, filtered, and dried under controlled conditions of temperature and humidity. The esters are mixed with plasticizers, dyes, and pigments and processed in various ways, depending upon the form of plastic desired.

These plastics may be fabricated by compression molding, injection molding, or continuous extrusion. Compression molding involves a temperature of 290 to 350° F. and a pressure of 2,000 to 5,000 pounds per square inch. The material is cooled in the mold under pressure so that it can be handled without deformation when it is discharged from the mold. In injection molding, the material is heated to the temperature at which it will flow easily under a high applied pressure. The hot plastic is forced into a cold mold and the finished piece may be discharged from the mold immediately without further cooling. The important properties of cellulose acetate include mechanical strength, impact resistance, transparency, colorability, fabricating versatility, moldability, and high dielectric strength. Some of its uses are as automobile steering wheels, airplane cockpit closures, electrical appliances, protective goggles, dials, control boards, and musical instruments.

Some of the noteworthy properties of *cellulose acetate-butyrate* are low moisture absorption, high dimensional stability, excellent weathering resistance, high impact strength, availability in colors, and improved finish. This plastic is used for exterior automobile lenses, furniture, refrigerators, hardware, gunstocks, and radio cases.

**Ethyl Cellulose Plastics.**—The base of these plastics is ethyl cellulose, a cellulose ether in which ethyl groups have replaced the hydrogen of the hydroxyl group. Commercial ethyl cellulose contains between 2.4 and 2.5 ethoxy groups per glucoside unit of the cellulose chain. The ethers are made by treating cellulose, such as wood pulp or cotton linters, with a concentrated solution of sodium hydroxide. The alkali cellulose formed is alkylated with such agents as ethyl chloride or ethyl sulfate. The alkylation must be carried out under carefully controlled conditions to prevent degradation of the cellulose chain and destruction of the alkylating agent. After the reaction is complete, the excess reagents are easily washed out. The ether is purified by washing it free from soluble materials. Ethyl cellulose is fabricated by injection molding or continuous extrusion. Some of its outstanding properties are unusually good low-temperature flexibility and toughness, wide range of compatibility, stability to heat, thermoplasticity, and electrical resistance. It is often used for protective coatings, furniture, extrusion wire insulation, injection plastics, and rigid containers.

### Thermoplastic Resins

**Acrylic Resins.**—The esters of acrylic and methacrylic acids are of more importance at present for plastics than other derivatives of these acids that also form resinous products. The formula of acrylic acid is

$$CH_2{=}CH{-}COOH$$

and the formula of methacrylic acid is

$$CH_2{=}C(CH_3){-}COOH.$$

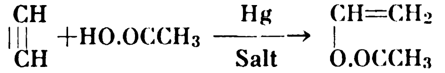
These esters of acrylic acid are made through treating ethylene chlorohydrin with sodium cyanide. The cyanohydrin that is formed is converted to the ester by combined hydrolysis and esterification in the presence of concentrated sulfuric acid and the appropriate alcohol. The esters of methacrylic acid are prepared by converting acetone cyanohydrin into esters of $\alpha$-hydroxyisobutyric acid and then dehydrating the hydroxyester. The esters of these acids are polymerized under the influence of heat, light, oxygen, or peroxides (benzoyl peroxide).

This material may be fabricated by casting, compression molding, injection molding, or continuous extrusion. In casting, the monomers are polymerized by heat in the presence of a catalyst. Compression molding temperatures vary between 285 and 360° F., and the pressures are between 1,500 and 5,000 pounds per square inch. Since these resins are thermoplastic, they should be cooled in the molds under pressure. For injection molding the conditions are 325 to 425° F. and 3,000 to 30,000 pounds per square inch. Cast sheets and cast extruded bars and rods may be shaped to order at temperatures of about 180 to 250° F. by the use of simple forms.

Outstanding properties of the acrylic resin plastics include superior transparency, dimensional stability, edge lighting, unlimited color range, water resistance, rigidity, and weather resistance. Some of the more important uses are for illuminating instruments (medical, surgical, and industrial), laminated glass interlayers, airplane windshields, dentures, artificial jewels, and furniture. Large amounts of the clear methyl methacrylic resin are employed for the eyepieces of gas masks, for watch glasses, and particularly for the transparent nose and blister windows in bombers and other airplanes.

**Vinyl Resins.**—The polyvinyl resins are synthetic materials made from compounds having the vinyl (—$CH{=}CH_2$) group. The most important members of this class are polyvinyl acetate, polyvinyl chloride, the copolymer of the acetate, and the chloride that combines the best properties of each one.

Vinyl acetate is a clear liquid to which a copper salt is added to the monomer to prevent polymerization.



$$\overset{\text{CH}}{\underset{\text{CH}}{|||}} + \text{HO.OCCH}_3 \xrightarrow[\text{Salt}]{\text{Hg}} \overset{\text{CH=CH}_2}{\underset{\text{O.OCCH}_3}{|}}$$

This may be left behind by distillation. The monomer, a catalyst such as acetyl peroxide, and a solvent are placed in an autoclave, and some heat is applied. After a short induction period the polymerization begins and is allowed to proceed to a predetermined limit. Then the unreacted monomer and the solvent are removed.

**Vinyl Alcohol and Acetal Resins.**—These resins are made from polyvinyl acetate which is first hydrolyzed to yield polyvinyl alcohol and then condensed with aldehydes to give a group of resins (or acetals). The polyvinyl acetate is hydrolyzed under controlled conditions by either acid or alkali which replaces the acetyl groups with hydroxyl groups to yield polyvinyl alcohol.

This method of preparation is used because polyvinyl alcohol cannot be prepared by direct polymerization as the monomer is an unknown compound. Vinyl alcohol, $CH_2{=}CHOH$, is the enol form of acetaldehyde, $CH_3CHO$, and is not stable. Polyvinyl alcohol is a unique plastic because it is plasticized by water and is completely soluble in an excess of water. The polyvinyl alcohol reacts with an aldehyde under the influence of heat and in the presence of an acid catalyst, such as sulfuric or hydrochloric acid, and the result is a typical acetal.

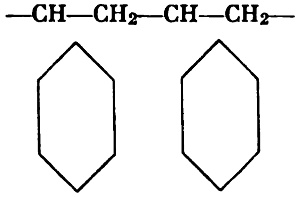
**Vinylidene Chloride Resins.**—This class of resins is one of the recent additions to the synthetic resin family. Based on unsymmetrical dichloroethylene, they were introduced in 1940 and may be formed by the polymerization of the monomeric vinylidene chloride, $CH_2{=}CCl_2$.

The resins formed by copolymerization range from a flexible material having a softening point of about 70° C. to a hard, thermoplastic solid with a softening point of 180° C. Their higher softening point indicates a greater degree of crystalline character. If the fibrous crystals are not oriented, the vinylidene chloride resin has a tensile strength of 8,000 pounds per square inch, which is an ordinary value. However, if the crystals are oriented by drawing, tensile strength may be increased to about 60,000 pounds per square inch. These resins may be fabricated by compression or injection molding or extrusion, with the use of specially developed techniques and equipment to get accurate control of properties and shapes.

Some noteworthy properties of these resins are high tensile strength, high fatigue and abrasion resistance, toughness, resistance to water and chemicals, nonflammability, high dielectric strength, ease of machining, and colorability. They are used for subway car seats, chair seats, fish leaders, and corrosion-resistant pipe.

**Styrene Resins.**—Monomeric styrene is made by the pyrolysis of ethylbenzene, which is synthesized from ethylene and benzene. The factors that affect the polymerization of the monomer are the temperature and the purity of the styrene. Polymerization at moderate temperatures without catalysts produces resins which are of high average molecular weight and which impart high viscosity to their solution. If the temperature is increased and catalysts such as benzoyl peroxide, oxygen, or stannic chloride are added, the average molecular weight and viscosity tend to decrease.

There is some uncertainty about the mechanism of the polymerization of styrene. It appears highly probable that the process may occur in at least three steps, chain initiation, chain propagation, and chain termination, and that the second step is very rapid compared with the first. In the light of recent investigations the configuration of the polymer is thought to be in the following head-to-tail form:



$$—\text{CH}—\text{CH}_2—\text{CH}—\text{CH}_2—$$

The lower polymers (20,000 to 30,000 average molecular weight and below) are brittle and have low mechanical strength. The higher polymers are strong and tough. This material can be compression molded, but injection molding is preferred. The temperatures for injection molding are from 160 to 230° C. and the pressure, from 10,000 to 30,000 pounds per square inch. Some of the outstanding properties are low specific gravity, high index of refraction, excellent acid and alkali resistance, excellent water resistance, dimensional stability, low molding shrinkage, excellent molded finish, ability to carry light, excellent electrical properties, and transparency. Thus this resin is used for acid, base, and bleaching solution bottle closures, high frequency radio and television parts, jewelry, signs, combs, and novelties.

### Protein Plastics

**Casein Plastics.**—Casein plastic is the product of the reaction between casein and formaldehyde.

The dry process for the manufacture of the plastic is based on the plasticization of casein powder, containing about 40 per cent total moisture, by the simultaneous action of heat and pressure. The soft plastic is extruded as rods or tubes from screw cylinder presses such as are used in the rubber industry. If sheets are required, the rods are flattened in hydraulic presses to the desired size. The rods and sheets of soft plastic are then hardened in an aqueous bath of 4 per cent formaldehyde. The hardening period varies from three weeks to six months, depending on the size of the rod or piece. After hardening, the material is dried to a moisture content of about 8 to 12 per cent. The drying period is almost as long as the hardening period. Recent improvements, however, have greatly reduced the curing time. It has been found that, if alum is mixed with the water before extrusion, or if the casein powder is treated with aqueous formaldehyde, the hardening begins at such a rate that the extruded material can be worked into various shapes and then hardened in the formaldehyde bath for only a few days. More recently, a thermoplastic molding composition has been made of formaldehyde-hardened protein material that comes finished from the mold. Thermosetting molding powders can be made from protein materials by mixing the formaldehyde-hardened proteins with phenolic resins and wood flour. Such a protein may be oil-free soybean meal. The curing time is about the same as for the phenolic resins, and the strength is about the same. Some outstanding properties are colorability, nonflammability, ease of polishing, ease of machining, and low hygroscopicity. The chief use of these plastics is for buttons, but they also find some use as buckles, beads, and novelties.

Under proper engineering control the hazards to workmen in the plastic industry are insignificant. In the Los Angeles area there are a number of companies producing various plastics which have afforded the author an opportunity to observe the effects of exposure to the various substances. As one peruses the foregoing enumeration of chemicals used in the production of plastics, it becomes evident that the possibility of toxic reactions obviously exists. However, the industry, being aware of inherent danger from undue exposure to these substances, has carefully guarded the workmen. The one disease which is most common in this industry is a dermatitis, which may be either a true contact dermatitis with the dust from the solid material or a result of sensitization from the vapors of formaldehyde, phenol, or other chemicals. Dermatitis can be avoided by installation of good ventilation, exhaust hoods over ovens, or wherever the material is in a hot state. Local exhausts should be used to remove dust. Showers, individual lockers, change of clothing, protective clothing, and protective creams, as well as the enforcement of personal hygiene, are requisites. In two plants the author found that the use of harsh soap was a contributing factor in the incidence of dermatitis.

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# CHAPTER XXXVII

# SPECIAL INDUSTRIAL PROCESSES

"Of what trade are you" has been the sine qua non of occupational disease diagnosis ever since it was first propounded by Ramazzini. But in his day the variety of occupations was comparatively limited. Today, according to McCord, there are twenty-five thousand different ways to make a living.

The titles used to distinguish modern-day occupations in industry are so much jargon to the average physician. Rarely do these titles indicate the type of work involved. Furthermore, most industrial processes are not clearly understood by members of the medical profession nor is the physician likely to find any description of these processes in his own medical library. Therefore, to inquire "of what trade are you" will yield little information unless the interrogator has some knowledge of what goes on within industrial plants.

It is the purpose of this section to provide the average physician with a description of common industrial processes which would otherwise not be available to him. Most of these descriptions are concise, although adequate space has been allotted to a few procedures which are of special importance. The intention is not to make a welder or a plastic molder out of the physician, but it is rather to help the physician understand these various trades.

## Electroplating

Electroplating is an exceedingly common method of producing protective metallic coatings. Since the advent of chrome plating many new developments have occurred in this field, and today the electrodeposition of various metals and alloys is being utilized.

In general, the principles of electroplating are as follows: After a careful cleaning, the article to be plated is made the cathode of an electrolytic cell, *i.e.*, the plating bath, which is composed of a solution of the salt of the metal, the coating of which it is desired to electrodeposit. The anode of this cell is the metal which it is desired to electrodeposit, but it may be another metal or a highly conductible material which is insoluble in the plating bath under the conditions of operation. With the passage of an electric current through this cell, metal ions enter the solution by dissolution of the anode, and these ions and identical metal ions of the electrolyte move toward and reach the cathode and are there deposited in the metallic state.

The electroplating process involves the use of:

1. Acid solutions

   (a) Acid copper consisting of copper sulfate and sulfuric acid to which small amounts of organic "additional agents" are commonly added.

   (b) Acid zinc baths consisting principally of zinc sulfate with additions of ammonium or aluminum chloride and organic agents; in this process ventilation is required although the zinc salts and the diluted sulfuric acid are not highly toxic.

   (c) Nickel plating is done in slightly acid baths; neither the nickel salts nor the chlorides or boric acid present are toxic although the nickel salts may be irritating to the skin and mucous membranes.

   (d) Acid lead baths; these consist of fluoborate, fluosilicate, perchlorate, or sulfamate of lead. There is little hazard except to the lead if such compounds are permitted to dry and form a dust.

2. Oxidizing acid solutions

   Chromic acid is the principal constituent of such baths and has a specific action upon the skin and mucous lining of the nose.

3. Alkaline baths

   The principal strongly alkaline bath (other than cyanide) used in plating is the alkaline tin solution; this consists principally of sodium stannate, containing sufficient free sodium hydroxide to prevent hydrolysis.

   Neither the stannate nor the sodium hydroxide is very toxic, but inhalation of any alkaline spray causes irritation and inflammation of nasal tissues. Moderate ventilation should therefore be provided for such processes as a safeguard against even occasional sprays.

4. Cyanide baths

   All of the cyanide baths, including those for copper, zinc, brass, bronze, cadmium, silver, and gold plating, contain (a) a soluble double cyanide of the metal plated, with either sodium or potassium; (b) some "free" sodium or potassium cyanide; and (c) in some cases, *e.g.*, zinc baths, free sodium hydroxide. The copper baths are frequently operated at high temperatures, e.g., 70° C. or 158° F., but most of the others are used at room temperature.

   From all such baths there is a constant slow evolution of the very toxic hydrocyanic acid, which is more rapid if the baths are heated. This gas arises from the action of carbon dioxide of the air to form sodium carbonate hydrocyanic acid. This reaction is retarded by the addition of free alkali to the baths. Except with heated baths, if good, normal room ventilation is used, the concentration of the hydrocyanic acid in the surrounding air is not likely to be sufficient to cause permanent injuries.

   The most serious hazard with cyanic solutions is the possibility that *acid* may be introduced and cause evolution of large volumes of hydrocyanic acid. Fortunately, careful precautions are taken against this possibility, so that very few instances have been reported.

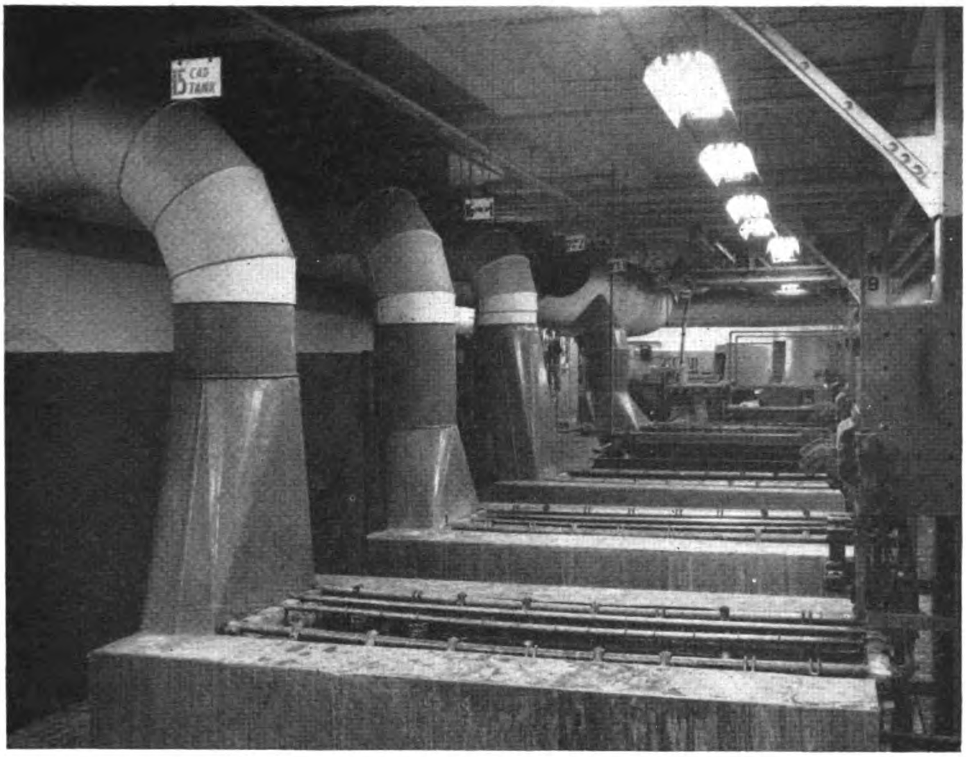

Fig. 81.—Cadmium- and zinc-plating tanks showing lateral exhaust.

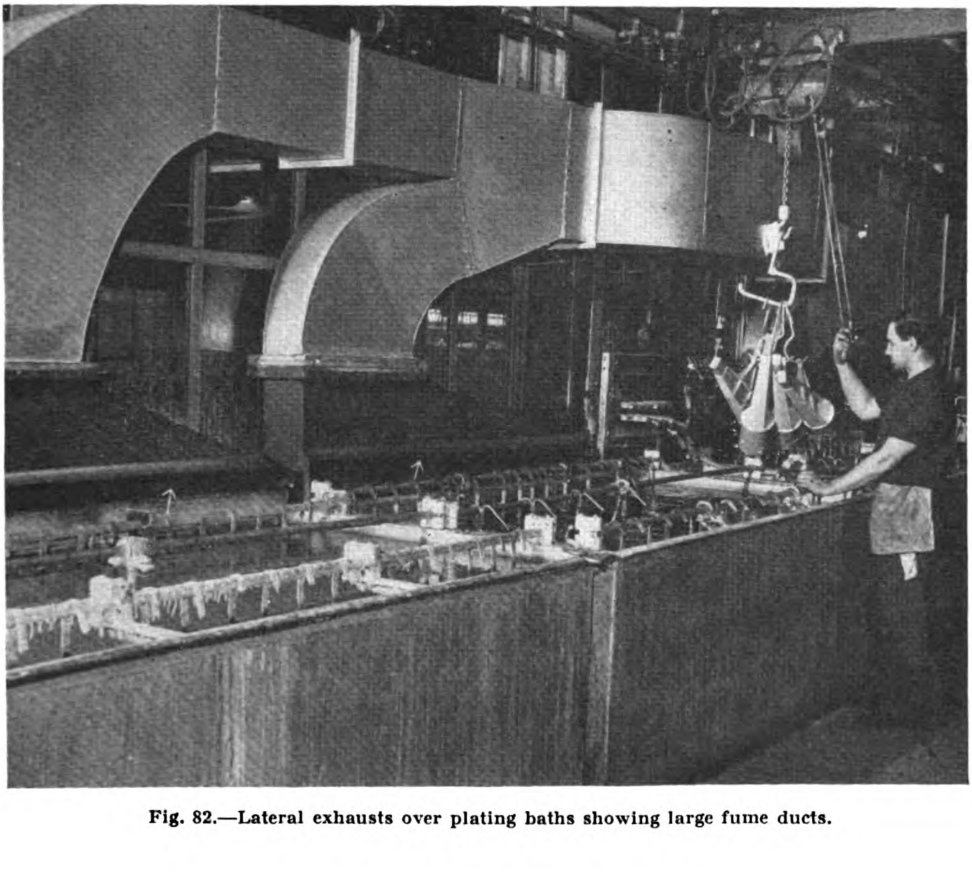

Fig. 82.—Lateral exhausts over plating baths showing large fume ducts.

> Most cyanide baths operate with efficiencies less than 100 per cent, and hence gases are evolved that carry spray into the air. For all such baths, special ventilation is desirable, though usually not so much ventilation as for chromium plating, in which latter process much higher current densities are used and hence more gas is evolved from a given electrode area. In silver and gold plating from cyanide baths, the volume and the current densities are usually low and the efficiencies high, so special ventilation is not generally required.

The engineering control measures include enclosure or partial enclosure of the tanks, mechanical exhaust system, and slot-type ventilation at the upper edge of the tank. The tanks and the exhaust system should be acid resisting (Figs. 81, 82 and 83).

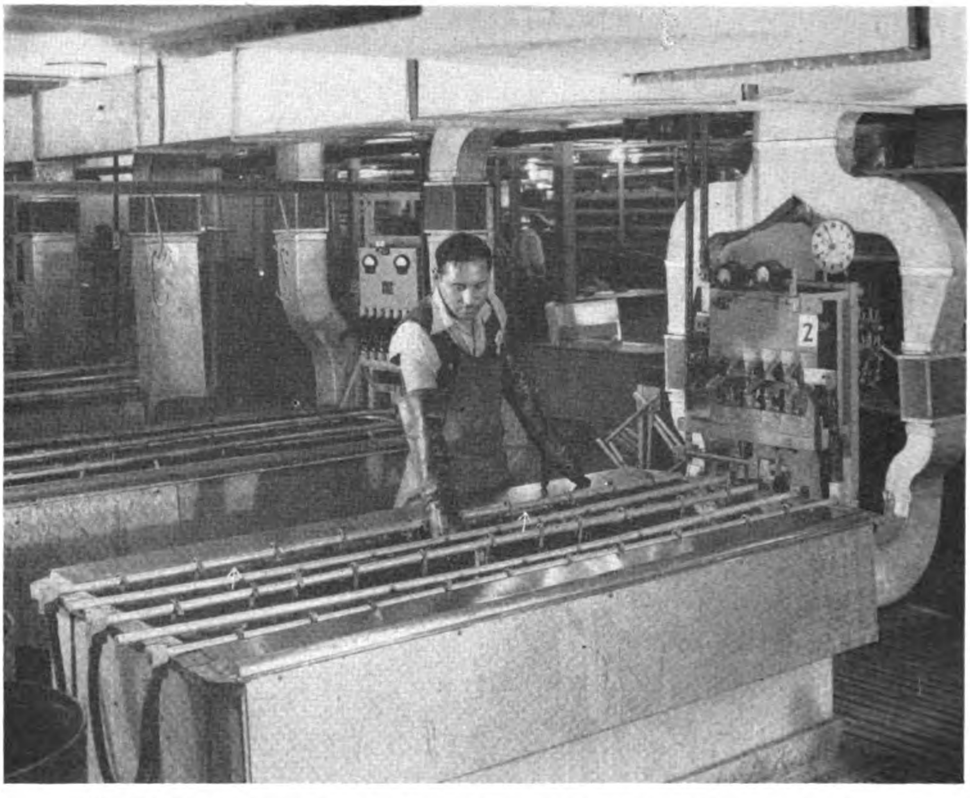

Fig. 83.—Lateral ventilation system installed on cadmium- and zinc-plating tanks.

If engineering control measures are followed, the electroplating process is comparatively safe. But, as in all other industries, violation of these safety rules may result in unwarranted injury to the workmen. A lamentable example of this occurred in Los Angeles in February, 1947, when one of the most disastrous industrial accidents in the history of that city took place. In this particular plant an explosion killed fifteen persons and injured nearly one hundred others due to the experimentation with perchloric acid in the electroplating process.

Perchloric acid, which has the composition $HClO_4$, is not a simple substance but an equilibrium mixture of the acid with small quantities of $Cl_2O_7$ and water, which forms a hydrate with the perchloric acid. The free anhydride contained in the acid is the cause of its lack of stability, for it gradually decomposes into chlorine dioxide and oxygen. The oxygen escapes and the $ClO_2$ remains in the liquid state. When this slow decomposition has proceeded for some time, there arises the likelihood of explosion.

## Anodic Treatment

The anodic treatment has been developed primarily to help aluminum and aluminum alloys to withstand corrosive agents. It is also known as the "aluminite process."

When aluminum, or an aluminum alloy, is made the anode in a suitable electrolyte, it becomes coated with a uniform, fine-textured, hard and adherent oxide film. The structure of the film contains fine pores, allowing the passage of the current, so that theoretically the process may be continued to produce a film of any required thickness, but according to tests on the thickness of film in relation to breakdown, a practical limit exists, above which the pores do not allow of equal current density over the whole surface, the film consequently being no longer uniform.

The reaction at the anode is primarily an electrolytic oxidation of the aluminum. The chromic acid ion is not changed by the reaction but it does act as an intermediate oxygen carrier. (Any substance may be used as the electrolyte which contains oxygen in the anion and is itself in its highest stable state of oxidation. However, the character of the oxide film is affected by the type of electrolyte.)

A great deal of heat is generated during the process (enough to melt a .040-inch sheet if no heat were given up to the electrolyte). This heat is rapidly dissipated into the surrounding solution which must be cooled during heavy runs (Figs. 84 and 85). There is some question as to whether the initial coating is aluminum oxide or aluminum hydroxide (the latter results from the addition of water, chemically, to the former). The weight of evidence favors the view of aluminum hydroxide formation with a slow change to the oxide occurring on exposure of the film to air. This view better explains the initial softness and absorptiveness of the film which are followed, on exposure, by hardening and loss of absorptive powers. (Aluminum oxide is infinitely harder than the hydroxide.) As stated, there are many electrolytes capable of use in the anodic process. However, the only other one of commercial importance used is sulfuric acid. The anodic process using this acid is known as the *alumilite* treatment. This treatment has the disadvantage of employing a highly corrosive acid and requires greater caution than in the case of chromic acid. However, it is capable of being controlled through wide ranges of voltage and electrolyte concentration to produce coats that vary from soft, very highly absorptive films to extremely hard films. Thus, the process is used where subsequent coloring or sealing with

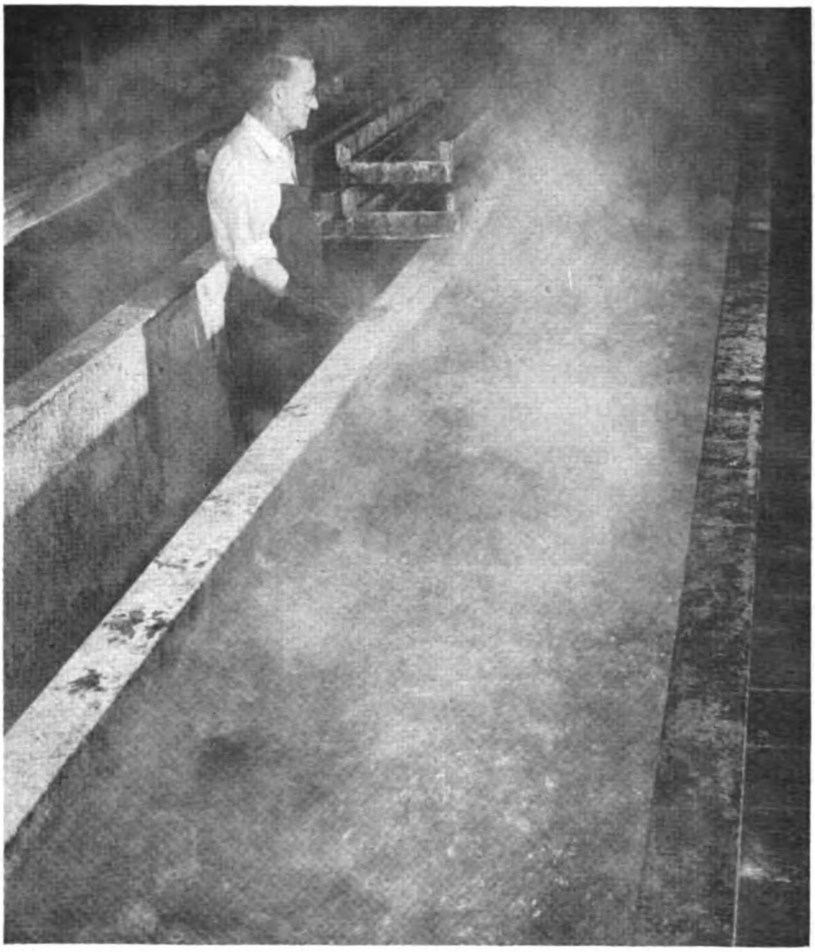

Fig. 84.—Anodic tank with no ventilation.

corrosion-inhibiting compounds is desired and also to produce wear-resistant surfaces.

The chromic acid process consists, as previously explained, in the electrolytic oxidation of the surface of the aluminum or aluminum alloy by treatment as anode in a solution of chromic acid. The oxide or hydroxide is formed as a film on the aluminum or alloy anode by gradually raising the applied potential to 40 volts. (The maximum value to which the voltage may be raised without causing breakdown of the film depends upon the composition of the alloy and other factors.) The control measures are essentially those used in electroplating.

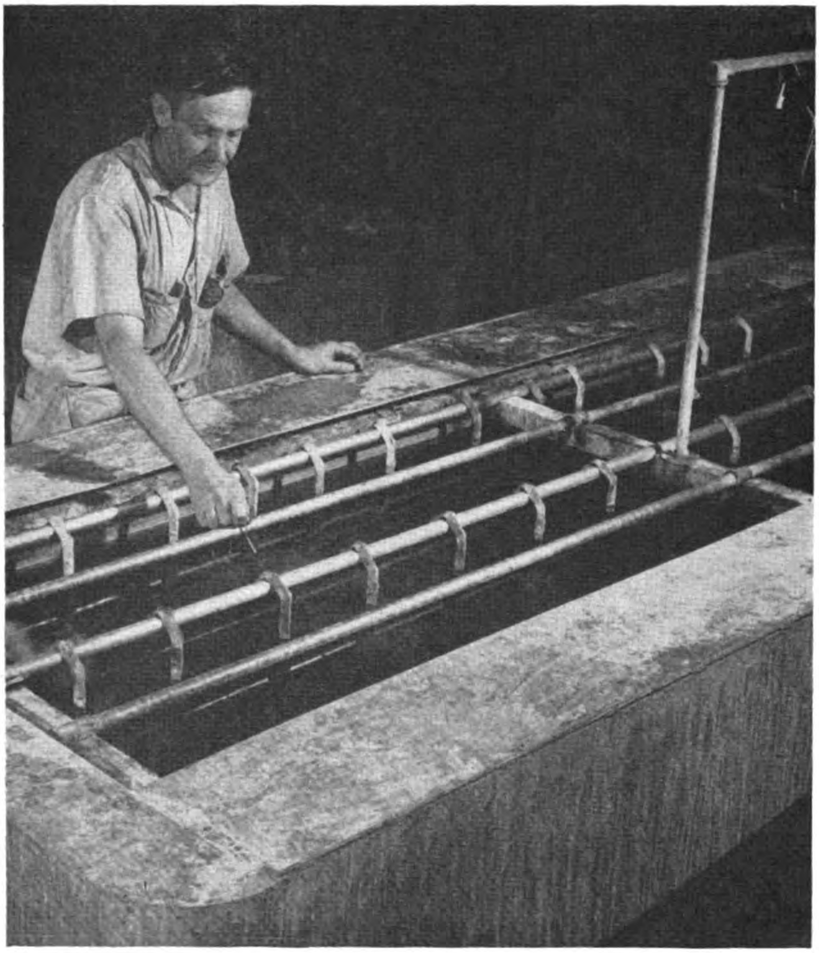

Fig. 85.—Same tank as shown in Fig. 84 with vents drawing off vapors.

## Degreasing

The oil and grease film which forms on the surface of metal objects must be removed before the parts can be successfully plated, enameled, or painted. There are several methods of doing this, but the most widely practiced method at this time is that of degreasing by means of a solvent. Carbon tetrachloride, ethylene dichloride, and trichlorethylene are the three solvents that have been found advantageous, and of these trichlorethylene is the one which is safest.

The utilization of trichlorethylene in degreasing depends principally on its solventability on oils and greases. This substance has the advantage of being noninflammable and nonexplosive.

Trichlorethylene is a heavy, low-boiling, colorless liquid with an ethereal odor. The following are the most important physical and chemical properties of the solvent:

| | |
|---|---|
| Formula | $CHCl\text{-}CCl_2$ |
| Molecular weight | 131.4 |
| Specific gravity 20°/4° C. | 1.462 |
| Boiling point (760 mm.) ° F. | 188.1 |
| Freezing point, ° F. | -126.3 |
| Latent heat of vaporization B.t.u./16°F. | 103.1 |
| Specific heat (liquid) B.t.u./16° F. | 0.223 |
| Vapor density (st B.P. and 760 mm.) | 4.53 (Referred to air 1.00) |
| Solubility in water at 25° C. (Gm./100 Gm.) | 0.17 |

Trichlorethylene vapor is stable on heating up to 257° F. (125° C.), but continued heating above this temperature in the presence of oxygen causes decomposition with corrosive acid formation (HCl). To inhibit such decomposition it is customary for the commercial solvent to contain small quantities of antioxidants. Such inhibitors also prevent decomposition due to light.

Although trichlorethylene is noninflammable, at high temperatures its vapors may decompose with the formation of corrosive and toxic substances, such as hydrochloric acid and phosgene. To prevent inhalation of these, the combustion products from open flames must, therefore, be vented so as to be disseminated safely outside of the building, and smoking in the vicinity of equipment using trichlorethylene must be prohibited.

Trichlorethylene may also be decomposed on prolonged heating in the presence of finely powdered aluminum. This is due to the catalytic effect of aluminum chloride that is first formed. Because of this, whenever there is aluminum dust or small chips on the parts to be cleaned, frothing may occur with accompanying bad odors, both indicative of decomposition. The addition of water to the trichlorethylene in the first wash tank as an extreme measure will prevent such decomposition when cleaning aluminum parts.

In the operation of a trichlorethylene degreasing tank, the solvent is heated to the boiling point with the consequent generation of concentrated vapor in the tank over the boiling liquid. The parts to be cleaned are dipped into the liquid and then suspended in the vapor zone for rinsing before withdrawing from the tank. A condenser built into the tank walls cools the vapor (Fig. 86), causing it to condense and return to the liquid supply, thus utilizing the solvent over and over. A well-adjusted balance between heat input, cooling capacity, rate of input of metal, and cleanliness of the solvent is required in order to insure the continuous operation of the cycle without escape of excessive quantities

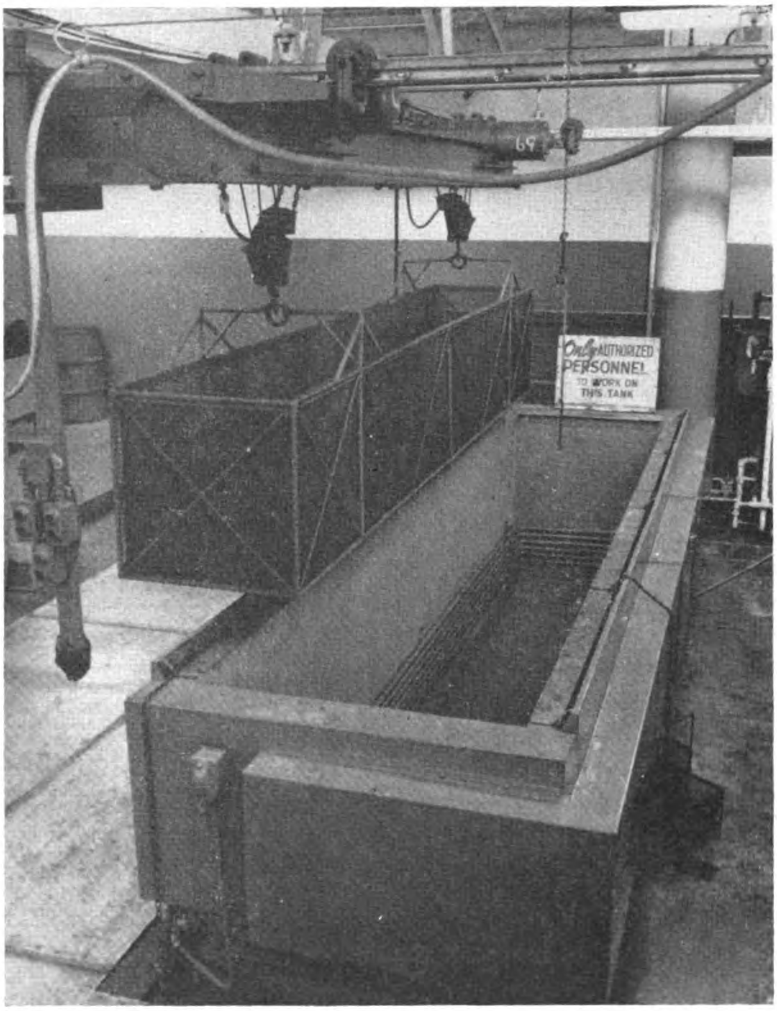

Fig. 86.—Degreasing tank. Note cooling coils on the inside of the tank to condense vapor. Lateral slot ventilation is also shown.

of the vapor from the tank. The constant generation of vapor, however, establishes a potential health hazard which becomes real when the operating balance between the liquid and the vapor is not constantly maintained. Because the loss of trichlorethylene vapor from the tank represents an increase in separating cost, the matter of proper operation assumes economic as well as hygienic importance and is thus more likely to be watched.

Small manufacturing plants, however, may and often do carry out trichlorethylene degreasing in makeshift equipment. In one case of ours, where a workman suffered severe intoxication, he worked over a large open metal tub under which there was a fire. This container was located in the middle of the room and away from any type of ventilation and was devoid of any pretense at exhausting the vapors. It is important for the industrial physician to inquire as to the design and make of degreasing tanks.

As recommended by Harris and associates[1], the tank location must also be selected to avoid disturbing drafts created by open windows and doors, fans, etc., since the action of such drafts is to drag vapor out of the tank and to disperse it in the room. In this connection, the location of the tank must be carefully considered in relation to paint spray booths which are commonly operated in conjunction with degreasing equipment. Spray booths require high rates of ventilation and strong air currents are thus established in the immediate neighborhood. Drafts may be created by the general ventilating system. To prevent this, air inlets should be located near the ceiling, remote from the tank, and provided with upward deflectors while outlets should be placed near the floor since the heavy vapor tends to gather at low points.

A gas-heated degreasing tank with natural draft ventilation of the combustion chamber should not be located in a room in which a mechanical exhaust system is installed unless ample free opening is provided for the intake of make-up air for the exhaust system. Otherwise a negative pressure would develop in the room which might cause a reversal in the direction of air flow through the natural draft vent pipe and thus bring the flue gases into the room. These gases may include not only the toxic products of gas combustion, but also phosgene resulting from the pyrolysis of trichlorethylene. The escape of trichlorethylene vapor into a room in which welding and similar high temperature operations are going on also presents a potential phosgene hazard and must be avoided.

The exhaust ventilation should be applied laterally at the edge of the tank, since the heavy vapor tends to roll over the edge and fall to the floor (Fig. 87). A ventilating rate of 60 c.f.m. per foot of tank perimeter has been found to be adequate. This produces an air improvement of approximately 20 feet per minute toward the exhaust openings at a distance of one foot from the slot. The application of ventilation to the tank disturbs the vapor zone in the tank somewhat and increases the solvent loss. This loss obviously increases with slot velocity. Moreover, it is known that the efficiency of control by lateral ventilation at a given total rate of air flow decreases with an increase in slot velocity. Hence, it is desirable to keep this velocity at a low figure. At the same time, the slot velocity must be high enough to make the entrance loss substantially greater than the internal loss in the section of duct into which the slot opens, so as to provide for uniform air flow over the tank area and into the exhaust system. An entrance velocity of 500 feet per minute appears to be the minimum practical value.

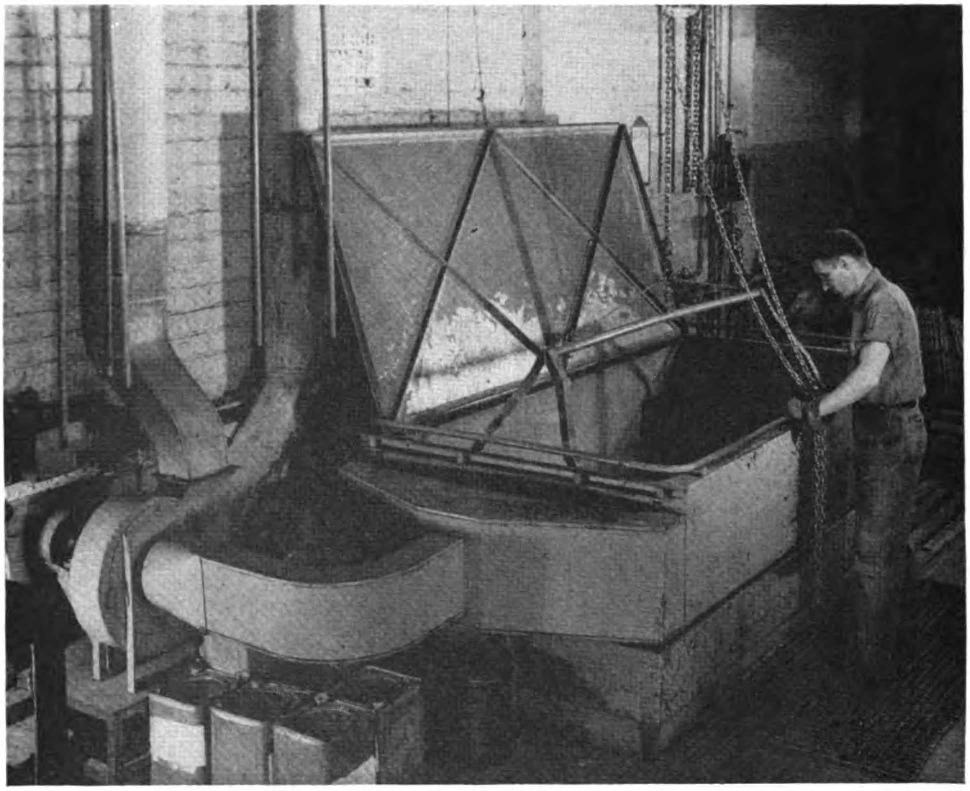

Fig. 87.—Ventilation on a degreasing unit. This tank has both cooling coils and lateral slot ventilation.

**Recommendations.—**

I. Design of equipment

A. Avoid the use of tanks and appurtenances other than of standard manufacture to insure proper design with respect to required balance between heat input, cooling capacity, and thermostatic control.

B. In every case provide a tank large enough to permit the complete immersion in the solvent of all parts to be handled, and wherever possible use an enclosed tank with automatic feed.

C. Provide a free-board distance from the maximum vapor level to the lip of the tank of not less than 15 inches.

- D. Provide a cleanout door for the removal of sludge.
- E. Provide thermostatic control of the heating and condensing zones and in addition provide safety thermostats to limit the solvent temperature and vapor height.
- F. For gas-heated tanks install a flue, from the combustion chamber to the outside, of adequate capacity and equipped with a proper diverter. The flue should be of acid-proof construction throughout its length.

II. Local exhaust ventilation

- A. All degreasing tanks in confined rooms (less than 20,000 cubic feet) and other installations not favorably located or properly operated must be provided with local exhaust ventilation. General ventilation is not recommended in place of local exhaust ventilation but may be required in addition.
- B. Exhaust ventilation should be by lateral movement into slots along the edge of the tank with a ventilating rate of 60 cubic feet per minute per foot of tank perimeter. The slot velocity should be approximately 500 feet per minute and provide for uniform ventilation over the entire tank area.
- C. Provide a ventilated booth or table where necessary for the temporary holding of cleaned parts for drying or for cooling.

III. Operation

- A. Operate the degreasing tank in accordance with the manufacturer's instructions.
- B. Pass work through the tank at a rate of 8 to 12 feet per minute.
- C. Keep tank closed when not in use.
- D. Clean the solvent and tank at proper intervals and otherwise follow manufacturer's instructions to avoid decomposition and loss of solvent.
- E. Provide solvent vapor absorbing type of respirators to be worn *during tank cleaning operations.*

## Pickling

Castings may be cleaned by sandblasting, tumbling, or pickling. Scale of various oxides, small clinging particles of metal, or particles of sand are removed by the acids used in the pickling process. These acids may be sulfuric, nitric, or dilute hydrochloric acids. Occasionally, but more rarely, hydrofluoric acid is used for a pickle. It is a strong acid, however, and may cause burns, as will nitric or sulfuric acid.

Pickling requires the use of special containers which are either earthenware or wood without nails. Perforated pickling boxes hold the material while in the acid. Upon removal from the pickling tank the castings are dipped into a tank containing hot water and soda. The soda neutralizes the acid. They are dipped next into a tank of plain hot water to wash off the soda scale or stain.

Due to the action of the acids and the various metals being cleaned, the formation of such gases as hydrogen sulfide, arsine, phosphine, and nitrogen oxides must be avoided or controlled. Injury from the various gases is avoided by complete or partial enclosure of the process, mechanical exhaust ventilation, and local slot exhausting. Workers should wear acid-proof clothing.

### Heat Treatment

Heat treatment is a combination of operations involving the heating and cooling of metals or alloys in the solid state. The purpose is to obtain desirable properties in the metal. The purpose varies according to whether the metal is cast iron, carbon steel, or such light metals as aluminum. The heat-treating process involves several steps which will be explained.

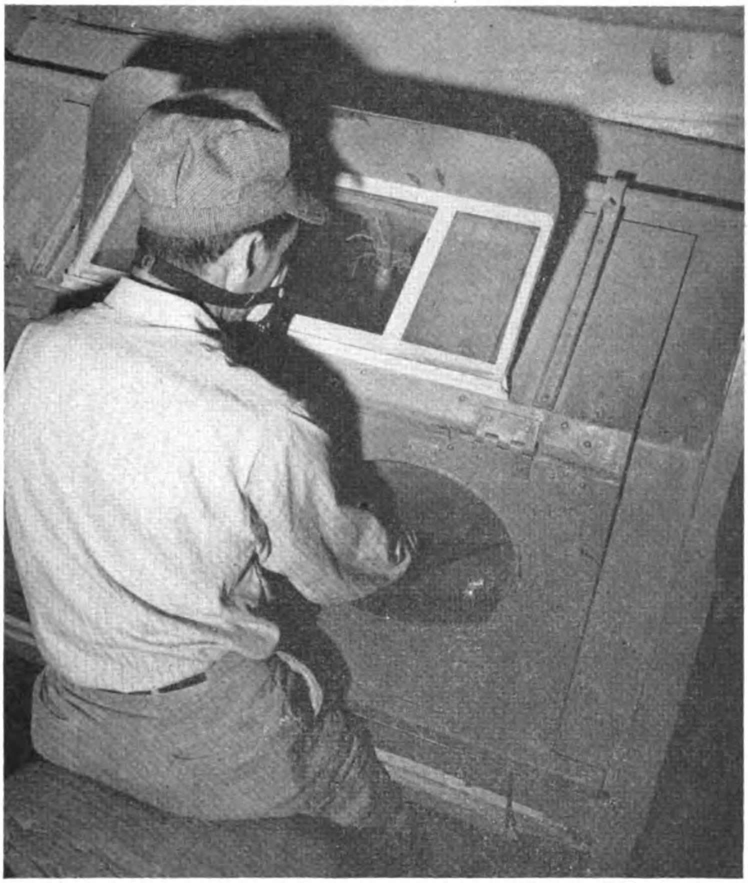

Fig. 88.—Enclosed unit for sandblasting. Note worker is outside of the operation and is additionally protected by respirator.

In the usual sense of the term, heat treating means the heating of metal to high temperatures and then the sudden quenching of the hot metal in water or oil baths. Annealing is also a heating and cooling procedure, but annealing usually implies relatively slow cooling.

The material to be treated is placed in the oven and subjected to a high temperature for variable lengths of time, this being predetermined by the nature of the metal, its thickness, etc. If it is to be suddenly quenched it is rapidly removed from the oven and dipped into a water bath or into an oil bath. In order to obtain case hardening, a carbon substance is sometimes packed on top of the metal held in the container when it is inserted into the oven. This "carburizing" may employ carbonaceous material such as charcoal or almond seeds.

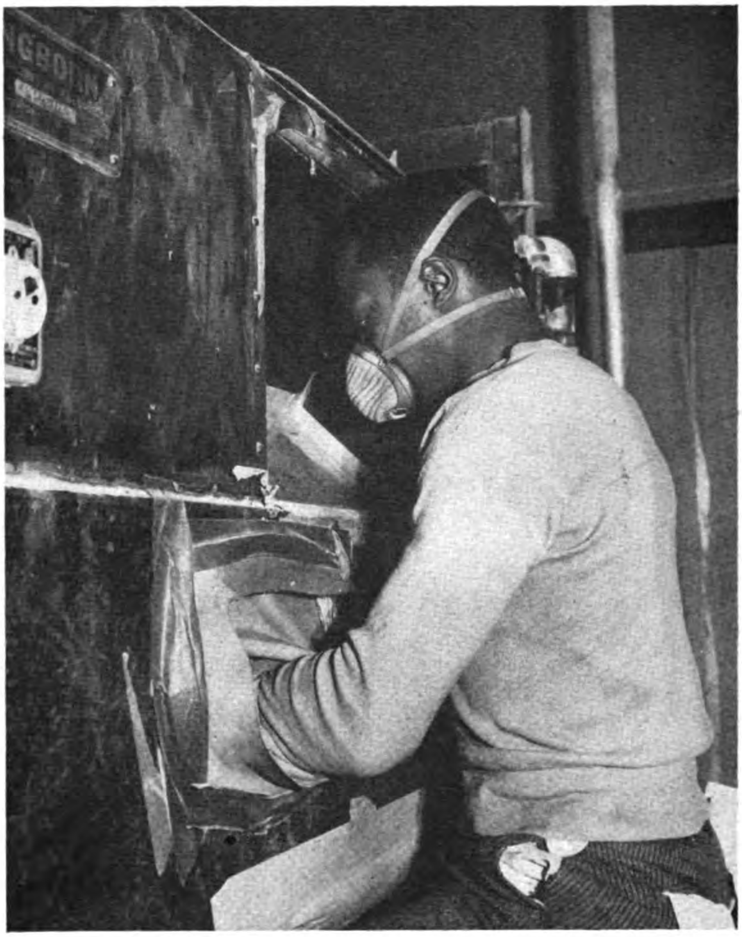

Fig. 89.—Sandblast operation similar to that shown in Fig. 88.

Surface hardening also may employ the use of cyanide, followed by a quenching bath. In this process the material usually consists of small parts which are suspended into the hot solution containing cyanide and then quickly removed to the quenching bath.

The hazards of the heat-treating process include possible exposure to carbon monoxide, cyanide, acrolein, and excessive temperatures. Exhaust fans, hoods, and ducts will control the carbon monoxide hazard as well as the cyanide hazard. Acrolein results from the action of heat on vegetable oil. This can be eliminated by substituting mineral oil.

### Sand Blasting

Under air compression, sand particles are propelled at a high rate of speed and strike metal surfaces, with the result that they remove scale or other particles left from the heat treatment. Sand blasting provides an excellent base for electroplating or painting. Because of the silica content of the sand the procedure must be carried out under severest control methods, such as exhaust ventilation, enclosed cabinets or rooms, personal protection devices, such as abrasive helmets fitted with supplied air respirators and, finally, by substituting shot blasting or tumbling for the sand-blasting method (Figs. 88, 89, 90*A* and 90*B*).

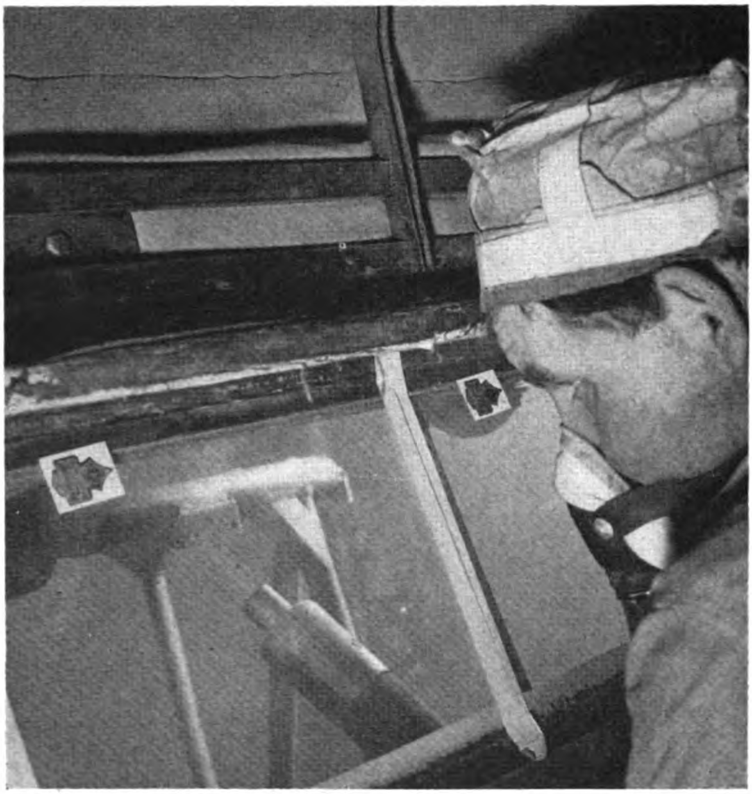

Fig. 90*A*.—Sandblast operation.

### Shot Blasting

Another method of cleaning castings, stampings, and forgings is by the centrifugal shot-blast machine. This machine does not use compressed air but hurls the steel shot radially from a wheel revolving at a very rapid speed. The hazards in this procedure are minimal, but methods must be used to care for the metallic dusts or silica dust that may enter the air from the surface of the metal in the process of being cleaned.

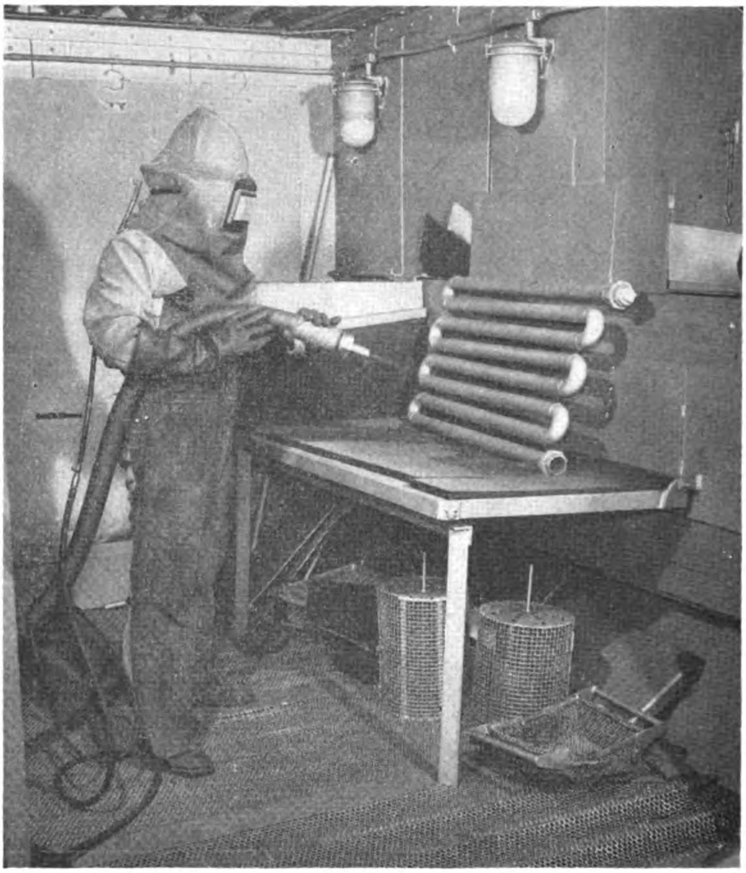

Fig. 90*B*.—Sandblast operation with operator protected by a hood and airline.

### Tumbling

Steel castings, brass, and bronze castings may be cleaned by tumbling. Into a revolving barrel containing slugs, carborundum, and ground slag, the metal to be cleaned is placed. In rolling, a form of tumbling, a portion of the barrel may contain an emulsion degreasing solvent. This method is often used where the metals may be covered by lubrication.

### Casting (Molding)

Foundry work involves a great many processes, the chief one of which is the making of castings. The largest and most important group of castings is made from the iron group of alloys, consisting of gray cast iron, malleable cast iron, carbon steel castings, and alloy steel castings. The nonferrous castings include those made of the copper base alloys, aluminum alloys, zinc base alloys, tin alloys, and lead base alloys. In recent years magnesium casting has become widely used.

The molds for the castings are of two general types, sand and metal. Molding sand is composed of two substances, silica and clay. Other substances found in the sand mixture are oxide of iron, lime, limestone, magnesia, soda, and potash. It is the silica content of the sand which makes foundry work a dangerous trade, unless it is carried on under controlled conditions.

From the flux and vapors which arise from the pouring of the metal into molds, workers may be exposed to hydrogen fluoride, fluoride salts, sulfur, and sulfur dioxide. Only the fluorides cause significant harm in this work. Magnesium burns have been mentioned in the discussion of that metal.

Foundry work demands extensive use of all types of exhaust systems, *i.e.*, air supply, general ventilation, and local exhaust ventilation. The shakeout and core knockout are procedures which are exceedingly dusty and must be submitted to adequate exhaust ventilation.

### The Ceramic and Pottery Processes

The ceramic industries, also called the clay product industries, manufacture chinaware, earthenware, stoneware, bricks of all types, enamels, enameled metals, and glass. They also manufacture a group of substances known as refractories which include firebrick, silica brick, alumina products, magnesite, and chromite brick.

The essential raw material used commonly in the ceramic industry is clay, feldspar, and sand. The clays are more or less impure hydrated aluminum silicate. In nearly all of the clays used, the basic clay mineral is kaolinite and, to a lesser extent, bentonite.

Of the feldspars used there are potash feldspar, soda feldspar, and lime feldspar, the first of these being the most frequently employed. Sand, the third chief raw material, is one chosen for its low iron content.

In addition to the raw materials there are a number of fluxing agents used, such as borax, boric acid, soda ash, sodium nitrate, fluorspar, cryolite, iron oxides, antimony oxides, and lead oxides.

It is not necessary to describe in detail the various steps that go to make the finished product. By the enumeration of the substances in use it is obvious that the ceramic or clay products industry subjects its workers to two important exposures; namely, silica and lead. This same statement applies to the glass and enameling industries. The lead and silica exist in the dust of the raw material and the frit. Since workmen often state they are handling frit, and since doctors do not always understand the meaning of the word, a brief explanation is in order. In the clay products industry, frit is the ground-up fluxing agent which has been previously listed. In the glass industry the various raw materials are mixed in proper proportions and charged in a melting furnace at a heat around 2500° F. After the batch has been uniformly melted, it is allowed to pour from the furnace into a tank of cold water. The contact with the cold water in the quenching tank shatters the melt into millions of friable pieces called frit.

Hygienic measures are primarily directed toward control of the dust and personal protection of the workers to prevent inhalation of the dust. The factor of inhalation has also to be kept under control in the burning of the clay products. A rare hazard exists at the kilns where improper combustion of fuel may give rise to carbon monoxide.

### Diesel Engines

In the western states, practically all of the trucking is done by diesel-powered machines. Likewise, most of the locomotive power over the mountains is from diesel-fueled engines. This type of engine is not only utilized because of the great power of a diesel engine, but also because of the economy of operation.

Inquiries are often made by physicians as to the type of exposure that exists in the operation of diesel engines. There appears to be some mystery as to the type of fuel and the type of combustion. For that reason a brief description is given here.

A gasoline engine mixes its fuel with the air for its combustion in the carburetor. The mixing is done before the fuel and air charge (the mixture) is drawn into the cylinder. If the proportions of the fuel and air are not right, the carburetor engine either will not fire at all or will smoke and fail to develop full power. There are other factors of good or bad operation, depending upon carburetor function.

The operation of a diesel engine is wholly different. The cylinder of the diesel always gets a full charge of pure air. The fuel charge is then sprayed or "injected" into the cylinder and mixes with the air *within* the cylinder itself. The amount of the cylinder air charge, and consequently the compression, does not vary from idling to full power. The only change as the load varies is in the amount of fuel injected into the cylinder combustion space. Control of the mixture is not necessary in diesel operation.

Another difference in the diesel from the gas engine is the absence of a spark to ignite the mixture of fuel and air. The diesel engine compresses gases within the cylinder to a high temperature, causing the mixture to ignite. Further details of operation are not necessary. The diesel engine is an internal combustion engine, capable of using a wide range of liquid fuels. In theory, the diesel engine can run on melted lard, cotton seed oil, or soya bean oil. But it operates best on what is known as "gas oils." In the distillation process the light distillates are motor gasolines, solvent naphthas, etc. The heavy distillates are heavy mineral oils, lubricating oils, and waxes. Between these there is a group of kerosenes, stove oils, and gas oils or "diesel oils." If the reader will recall the discussion on the chemistry of the hydrocarbons presented earlier in this book, he will note that the first in the series of gases was methane, with a formula of $CH_4$. The series progresses through ethane, propane, butane, and pentane. If one carries the series far enough, one comes to tetradecane, $CH_{14}H_{30}$, and hentriacontane, $C_{31}H_{64}$, which are the diesel oils.

Under ordinary conditions there are no more hazards residing in the operation of diesel engines than there are in gasoline engine operation. If, however, the gases from the exhaust collect in confined spaces, harm may arise. The constituents of these gases are carbon monoxide, the oxides of sulfur, and nitrogen, aldehydes, and carbon dioxide.

### The Fluorescent Lamp Process

A fairly recent development in the lighting industry is the use of certain inert gases such as argon and neon. These gases are chemically inert but possess desirable electrical properties.

The neon light is a "gas conductor" lamp consisting of glass tubing with electrodes sealed in each end and filled with neon, helium, or argon-helium mixtures, with or without mercury. The light is caused by the flow of electric current while the type of gas contained in the tube determines the color.

The fluorescent lamp is a tube filled with one of these rare gases and a drop of mercury, with an electrode at each end. The inner walls of the tube are lined with a fluorescent salt or "phosphor." These phosphors differ in their chemical consistency according to the color of light desired. The common phosphors are calcium tungstate, magnesium tungstate, zinc silicate, zinc beryllium silicate, cadmium silicate, and cadmium borate.

In the discussion on beryllium, mention has been made of the unusual reaction upon the lungs of those unduly exposed in the fluorescent light industry.

### Storage Battery Manufacturing Operations

- Grid castor or molder
  - Forms battery grids to be used in making storage battery plates by melting and pouring molten lead into cast iron molds
  - Operates melting furnace
  - Feeds pig lead to furnace
  - Controls temperature of metal and molds
  - Adjusts and coats molds as required and removes grids from mold

Lead parts molder
- Makes storage battery parts, such as cell connecting links, element straps, and posts, by pouring molten lead into iron molds
- Operates melting furnace and controls temperature of metal and molds
- Coats or powders and adjusts molds as required
- Feeds pig lead to furnace and removes castings from mold

Paste mixer
- Mixes lead oxide for storage battery grids on a mechanical mixer
- Measures specified amounts of lead oxide into mixing machine
- Mixes oxides dry and then adds water and acid and mixes wet
- Removes finished paste from machine
- Controls temperature and plasticity of paste

Pasting machine operator
- Fills storage battery grids with lead oxide paste
- Feeds grids and paste to machine
- Adjusts machine as required, controls thickness of plates, and feeds excess paste back to machine

Plate racker
- Racks, stacks, or dips pasted plates from pasting machine as required by specified process

Plate splitting machine operator
- Splits double pasted storage battery plates into single plates
- Feeds double plates to machine and removes single plates
- Adjusts machine to cut and clean plate lugs to required length

Element assembler or burner
- Assembles plates into battery elements
- Racks plates in element burning rack; places connecting strap on plate lugs in templer of burning rack
- Fuses straps to plate lugs with flame torch
- Removes elements from racks

Element grouper
- Assembles positive and negative elements with plates in proper sequence into storage battery cells

Element insulator
- Insulates positive plate from negative plate in storage battery cell
- Places elements in jig and inserts separator between positive and negative plates

Element cover placer
- Places battery cell cover on insulated elements

Element placer
- Places completed elements in storage battery containers in their proper sequence

Battery sealer
- Seals battery cell covers to container with melted battery sealing comopund
- Controls temperature of compound in heating kettle

Top connector or link burner
- Connects cells in battery container by fusing lead link with a flame torch to element posts

Terminal post builder

- Builds terminal posts on storage battery by placing an iron mold on element post and fills mold with molten lead, using a flame burning torch

Battery chargers

- Fill batteries with electrolyte and place on charge
- Determine gravity of electrolyte, rate and length of charge; control temperature during charge
- Test voltage and gravity of electrolyte to determine completion of charge

## Welding

The multiple purposes to which welding can be put makes it an indispensable aid to the one-man shop as well as to all large industries. Oxyacetylene and carbon arc welding have been used in this country for over thirty years. Electric resistance welding was invented by Thompson in 1886 but received its greatest usage during World War II in airplane construction.

**Oxyacetylene Welding.**[2]—Oxygen is a colorless, tasteless, and odorless gas. Its main characteristic, of course, is its ability to support and intensify combustion. Pure oxygen is a very active substance even at ordinary pressures. This activity is increased still more when the oxygen is at high pressure, and in this condition it may combine violently with oil or grease even at ordinary temperatures.

Oxygen for welding purposes is produced mainly from liquid air. Air consists of about one-fifth oxygen, the remainder being nitrogen, with a small percentage of rarer gases such as argon, neon, and helium. Air can be liquefied and then separated into its component parts by a carefully controlled process of rectification, which takes advantage of the fact that liquid nitrogen boils at a lower temperature than liquid oxygen. The oxygen is separated from the mixture and delivered to a storage holder. From there, it is compressed in cylinders for shipment.

Acetylene is a colorless, combustible gas and has a characteristic odor. It has a compound of carbon and hydrogen, having the chemical formula $C_2H_2$.

Acetylene forms explosive mixtures with air and oxygen. Regulations and laws forbid the generation, compression, or use of fuel acetylene at pressures above 15 pounds per square inch, for higher pressure may cause explosions under certain conditions.

Acetylene is produced by the chemical reaction between water and calcium carbide, $CaC_2$. When a piece of calcium carbide is dropped into water, bubbles of a gas that is acetylene gas will rise. The reaction is as follows:

$$\underset{\text{Calcium carbide}}{CaC_2} + \underset{\text{Water}}{2H_2O} = \underset{\text{Acetylene}}{C_2H_2} + \underset{\text{Hydrated lime}}{Ca(OH)_2}.$$

The calcium carbide is a gray stonelike substance produced by smelting lime and coke in an electric furnace. The hydrated lime is the whitish residue that remains in the water after the action has stopped.

**The Oxyacetylene Flame.**—The oxyacetylene flame, like all other flames, is a phenomenon produced at the surface where two gases meet and undergo combustion with the evolution of heat and, more or less, light. With commercially pure oxygen and acetylene, however, the hottest known flame may be produced, its estimated temperature being in the neighborhood of 6000° F.

The chemical reaction for the complete combustion of oxygen and acetylene is as follows:

$$\underset{\text{Acetylene}}{2C_2H_2} + \underset{\text{Oxygen}}{5O_2} = \underset{\text{Carbon dioxide}}{4CO_2} + \underset{\text{Water vapor}}{2H_2O}$$

This means that two volumes of acetylene and five volumes of oxygen combine, or react, to produce four volumes of carbon dioxide and two volumes of water vapor. In other words, for complete combustion of acetylene the ratio of oxygen to acetylene is 2½ to 1.

The flame temperature produced is dependent upon the relative proportion of these two gases. Since the flame temperature alone, however, is not the only factor in the commercial use of the oxyacetylene flame, varying ratios have been found most suitable for different purposes. For oxyacetylene welding and heating, the most suitable mixture is generally obtained by employing approximately equal proportions of oxygen and acetylene through the blowpipe. This mixture, when burned at the tip of a properly designed torch or blowpipe, produces what is known as a "neutral" flame because its action is neutral in effect, that is, neither oxidizing nor carburizing. In the combustion of this mixture, there is a double chemical action as follows:

$$\underset{\text{Acetylene}}{C_2H_2} + \underset{\text{Oxygen}}{O_2} = \underset{\text{Carbon monoxide}}{2CO} + \underset{\text{Hydrogen}}{H_2}$$

This means that one volume of acetylene for one volume of oxygen will produce two volumes of carbon monoxide plus one volume of hydrogen. This reaction takes place at the inner cone of the flame where the highest temperature is developed. The carbon monoxide and hydrogen are then burned to carbon dioxide and water vapor as follows:

$$\underset{\text{Carbon monoxide}}{2CO} + \underset{\text{Oxygen}}{O_2} = \underset{\text{Carbon dioxide}}{2CO_2}$$

$$\underset{\text{Hydrogen}}{2H_2} + \underset{\text{Oxygen}}{O_2} = \underset{\text{Water vapor}}{2H_2O}$$

The oxygen required for the burning of carbon monoxide and hydrogen to carbon dioxide and water vapor is provided by the atmosphere. It can be seen from the preceding that in the neutral oxyacetylene flame 1 part of oxygen is provided from the torch or blowpipe and 1½ parts of oxygen are provided from the atmosphere in order to complete the combustion of 1 part of acetylene (Fig. 91).

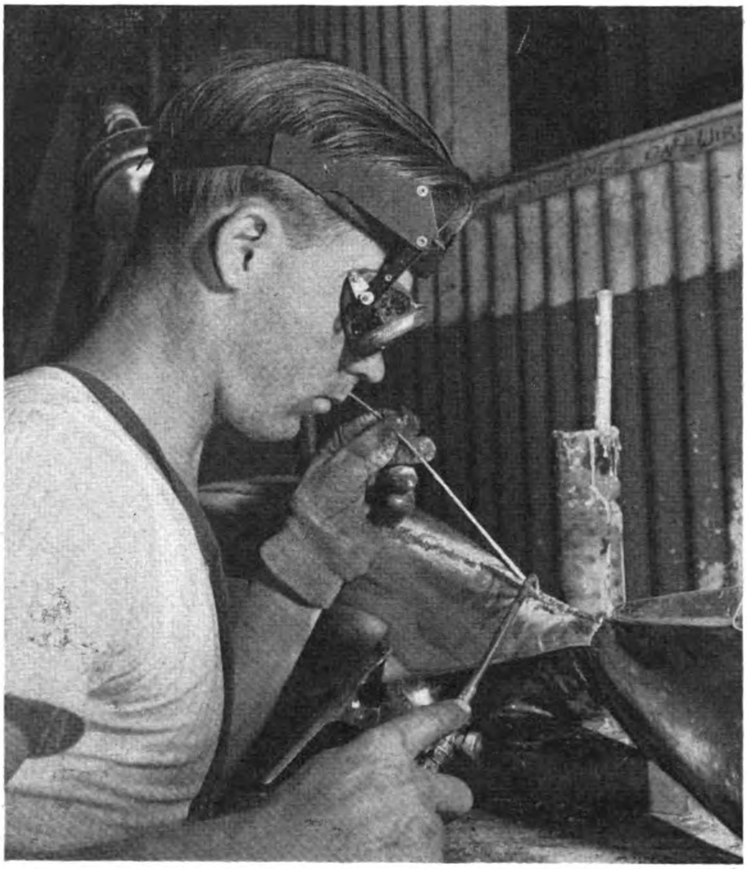

Fig. 91.—Welding by acetylene torch.

Oxyacetylene welding is a form of fusion welding wherein the parts to be welded may be fused together without the addition of material from the welding rod. Usually, however, in fusion welding a filler metal is added in the form of a rod. The material in these rods varies and will be mentioned subsequently under arc welding. During the process of melting the metals an oxide gathers on the surface of the parts to be welded. Since this oxide does not melt at the welding temperatures it prevents a smooth coalescence of the weld metal. A flux is therefore employed that melts somewhat below the welding temperature and breaks down the surface skin of oxide, thus permitting the liquid metal from the rod to flow into the molten parent material.

Oxyacetylene is also used for cutting metals. This is a process of preheating the material to be cut to its kindling or ignition temperature and rapidly oxidizing it by means of a closely regulated jet or stream of oxygen issued from a special tool called a cutting blowpipe or cutting torch.

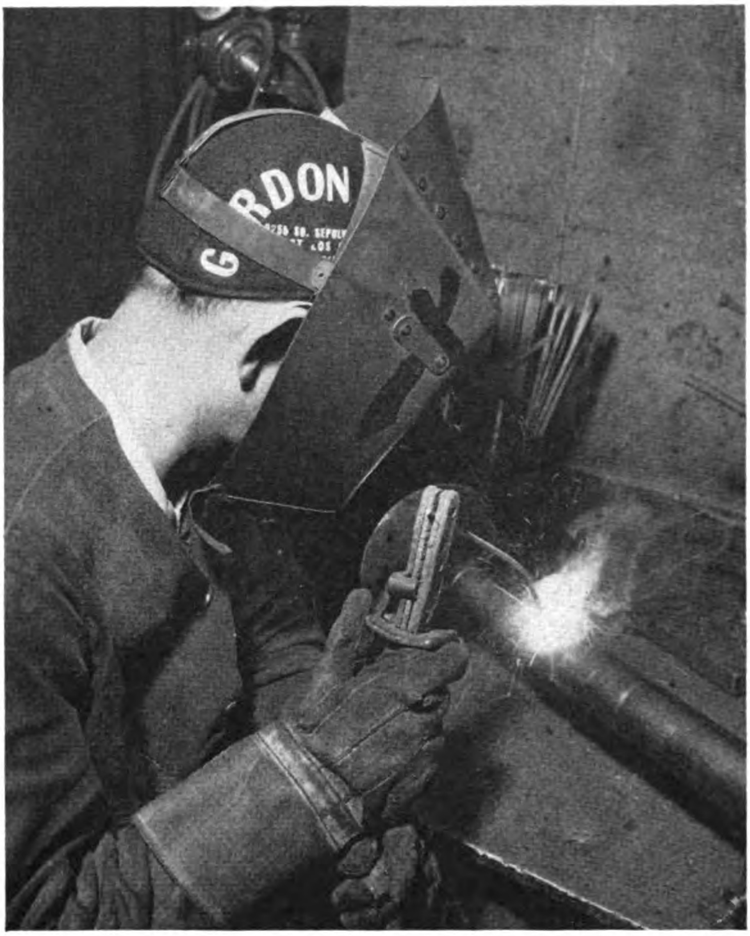

Fig. 92.—Electric arc welding.

**Electric Arc Welding.**—An electric arc consists of an incandescent stream of metallic vapors carrying an electric current. It is formed when two conductors of an electrical circuit are brought together and then separated while there is sufficient voltage available to maintain the flow of current through the intervening gaseous medium.

The heat liberated at the arc terminals and in the arc stream is used to melt the metals to be welded at the point of contact, so that they will flow together and form a solid integral mass. Thus, different parts may be joined, or material may be added to the surface of a metal.

The temperature of the arc is in the vicinity of 6500° F.; and when this tremendous heat is concentrated at point of welding, it melts a small pool of metal in the work. If any additional metal is required for the welding, it is obtained from a wire or rod, which is melted by the heat of the arc and deposited into this small pool in molten state. The molten metal in the pool is agitated by the action of the arc, and thus the base, or parent, metal and the added metal are thoroughly mixed and refined so that, after cooling, a sound union is formed.

The welding circuit consists of a welding machine, two leads, an electrode holder, an electrode, and the work itself (Fig. 92).

In the *carbon arc process,* the arc is formed between the work and a carbon rod. The heat of the arc melts a pool in the surface of the work to be welded; and additional metal, when required, is supplied by a separate metal filler rod which is melted in the arc. The carbon electrode is consumed, very slowly, owing to oxidation and vaporization only. Carbon arc welding practice is similar to welding with the oxyacetylene flame.

**The Metallic Arc Process.**—In the metallic arc process, the arc occurs between the work to be welded and a metallic wire which, like the parent metal, is melted by the intense heat of the arc. The melting wire, or electrode, thus furnishes the "filler metal" and is fed at a uniform rate toward the base metal.

**Shielded and Unshielded Arc Welding Processes.**—Molten steel has an affinity for oxygen and nitrogen. When exposed to the air, molten steel enters into chemical combination with the oxygen and nitrogen of the air to form oxides and nitrides in the steel. These impurities weaken and embrittle the steel and lessen its resistance to corrosion.

An ideal weld is one whose properties are equal or superior to those of the parts joined. With the arc welding process, such a weld can be obtained by effectively protecting the molten filler metal in the arc stream and the weld metal from the oxidizing and nitrogenizing effects of the air during the entire range of liquefaction and solidification.

An arc may be shielded by completely enveloping it with an inert gas that will not enter into chemical combination with the molten metal and that, at the same time, will prevent the molten metal from coming in contact with the atmosphere.

Arc welding processes, in which the molten filler, weld, and base metals are effectively protected, are classified as shielded processes to distinguish them from unshielded processes in which no protection or only partial protection is given.

Coated or covered-electrode welding includes all processes in which shielding is accomplished by combustion, vaporization, or melting of the several ingredients of the covering applied to the wire core of the electrode.

Taper-shielded welding includes all processes in which a ribbon or cord is independently and automatically fed into the arc zone and functions like the coverings on coated electrodes.

Gas-shielded welding includes those processes wherein the arc zone is surrounded by an inert gas supplied from an independent source.

Flux-shielded welding includes all processes in which a flux in the form of a paste or powder is placed in or at the point ahead of the arc and is wholly or partly vaporized, or melted, as the welding proceeds.

Bare-electrode welding, an unshielded process, includes the use of lightly coated electrodes as well as truly bare electrodes.

**Electric Resistance Welding.**—The fundamental principle of the electric resistance welding process resembles that of forge welding. In the latter process the workpieces are heated in a forge and then hammered together. In resistance welding, the workpieces are heated to a plastic state at a given junction by passing a relatively high current across the joint. They are then pressed together between the contacting terminals.

The welder consists mainly of a single phase transformer having a secondary of one or two turns. A low voltage is required between the terminals, and the amount of current needed is dependent only upon the size of the pieces to be handled. A heavy frame, built about the transformer, supports not only the clamping jaws to handle the work, but also the mechanical devices used to regulate the heat and the pressure. The electric current is carried to the work by heavy copper conductors, and the resistance to the flow of the current is thus concentrated in the work. This resistance causes the work to become heated enough to reach a "white heat" and to become plastic. The weld is then formed by cutting off the current and applying pressure to these plastic sections.

The union is as strong as any part of the pieces that have thus been joined. An outstanding feature of the process is that the heat is generated principally at the center of the parts being welded and in line with the points of contact.

Resistance welding differs from other forms of welding in that there is no introduction of any foreign material into the weld; the welding heat is definitely localized at the desired point, with resulting control of the extent of fusion; there is a high rate of production of welds; it easily unites difficult shapes which could not otherwise be easily joined, as in the case of fine wires and very light sheets; ferrous and nonferrous

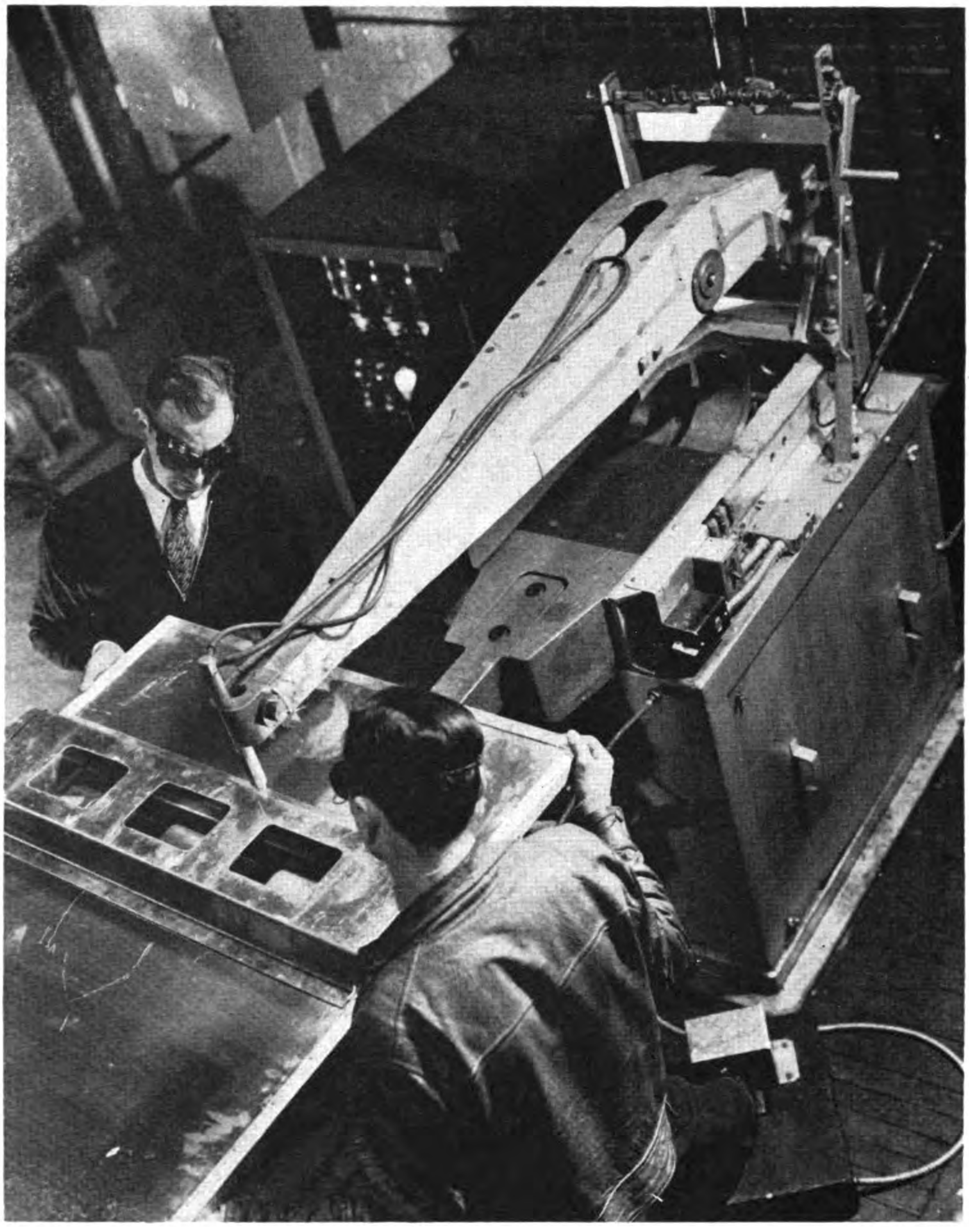

Fig. 93.—Spot welding, a sheet assembly with a.c. spot welding machine. (Courtesy Aluminum Company of America.)

alloys are readily welded; and even totally different materials, as copper and steel, can be welded together. It facilitates the application of automatic operation, for the equipment can be set up to give the desired results, and there is little skill required on the part of the operator. Also, all welds thus made have approximately the same characteristics.

The subdivisions of the resistance-welding process are determined by the location of the weld and the method of making the weld. They are principally spot, butt, and seam welding.

In spot welding the metals are welded or fused at a spot. Since it is usually applied in the joining of light sheets, a large current, at a low voltage, is discharged through two or more pieces of metal. The discharge of current is followed with sufficient pressure to unite the molecules of the workpieces. The current is cut off before the mechanical pressure is released so as to prevent burning the contacting electrodes (Fig. 93).

Simple spot welds are obtained when small contact areas on the electrodes are used. If, prior to welding, one or more points, or projections, are raised above the plane surfaces of the parts to be welded and if these parts are placed between electrodes having large contact areas, then the resulting weld is called a projection spot weld. Ridge welding is obtained if ridges are raised instead of point projections. The ridges of one part should cross the ridges on the other part or parts to be welded.

Butt welding is obtained when pieces having practically the same cross section are welded end to end. The weld is made by heating the entire areas of the butted surfaces to a fusing temperature and applying pressure. The pieces are held in alignment by suitable clamping electrodes, or jaws. If the pressure is applied and persists while the parts are being heated, the welds are characterized by large upsets or swelling at the fused section and are known as slow butt welds. If, while the power is applied to the clamping electrodes, the surfaces are brought close to one another so as to cause a spark, or flash, to jump across the gap until the proper fusion temperature is obtained and if the forging pressure is then suddenly applied, a flash weld results. It is marked by small upsets.

The seam weld is similar to a spot weld, except that circular rotating electrode wheels, contacting the opposite faces under pressure, are used to produce the effect of a seam. The weld may be continuous if the current is passed at short enough intervals, or it may consist of a series of spot welds if the current is passed at long enough intervals. Both lap and butt seam welds can be thus made (Fig. 94).

**Hazards of Welding.—**

1. ARC FLASH BURNS.—Excessive exposure to the welder's arc will produce actinic or ultraviolet conjunctivitis. This condition consists of swelling of the eyelids, photophobia, and a feeling of "sand in the eyes," together with injection of the eyeballs. The onset is usually delayed by some hours. The acute stage lasts for about twenty-four to thirty hours.

2. BURNS TO THE SKIN.—The unprotected skin around the neck, upper chest, arms, or side of the face may receive burns from the arc

or from the molten metal. The arc burns are comparable to sunburn. The skin burns from the metal are superficial as a rule, and both types are easily prevented by adequate protective equipment.

3. Fumes.—The inhalation of various fumes is always a hazard but rarely does an illness result from such exposure because of the control measures in vogue. Faulty combustion can produce carbon monoxide or nitrogen dioxide; the cutting of lead-coated material may result in lead poisoning, or the presence of combined metal oxides may cause metal fume fever.

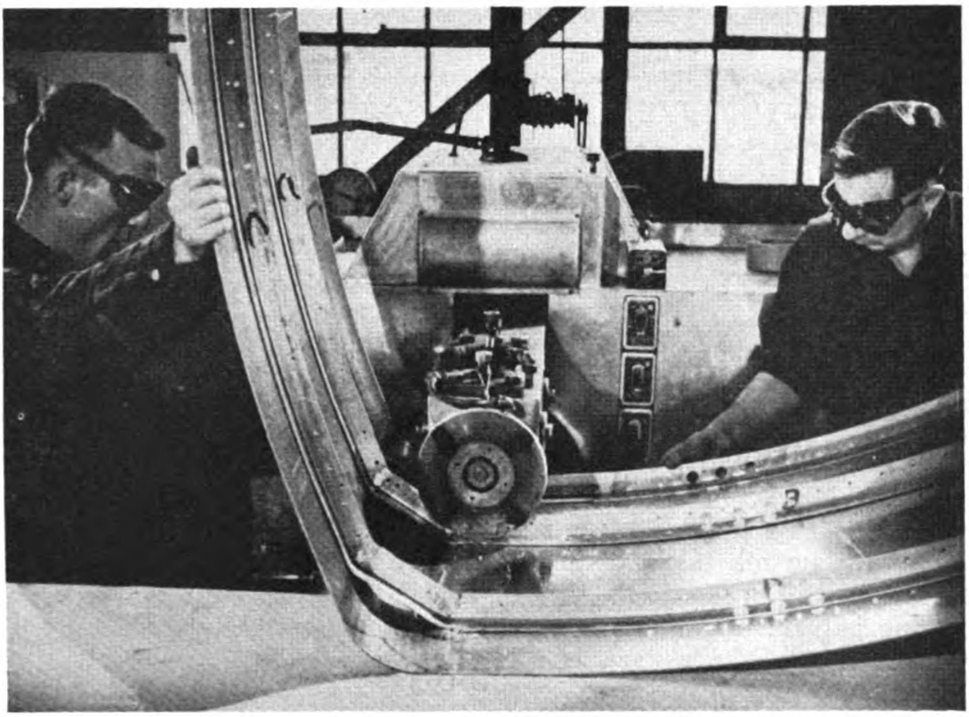

Fig. 94.—Intermittent seam welding in aluminum alloy gasoline tank. (Courtesy Aluminum Company of America.)

The prevention of illness from the inhalation of fumes or metal oxides resides in the control measures consisting of helmets or shields, local exhausts, respirators, and individual air-supplied masks. These will be described later.

**Composition of Welding Rods.**—The material to be found in welding rods is fairly well standardized. The composition varies according to the purpose and the parent material to be welded. An analysis of the rods in general use by Tebbens and Drinker[3] showed the mild steel rods were low in carbon, phosphorus, and sulfur. The stainless steels contained about 18 and 8 per cent of chromium and nickel. The aluminum electrodes were about 5 per cent silicon and the Everdur copper about 4 per cent silicon. Monel is approximately 70-30 nickel and copper, while Inconel is about 80-14-6 nickel, chromium, and iron.

In the mild steel coatings, manganese as ferro-manganese is usually used as a deoxidizer. Titanium dioxide is frequently incorporated as an arc stabilizer. Asbestos fiber or twine and other mineral silicates are often included, while sodium or potassium silicate is used for binding the mixture. Frequently sugar, wood pulp, or other carbohydrates are included to assist in producing a reducing atmosphere at the arc.

Coatings of the alloy and nonferrous rods become important because of the mineral anions. Inorganic fluorides and carbonates are common ingredients, while chlorides and borates are used less often. As in mild steel coatings, managnese minerals and water glass are usually present, but organic materials are seldom employed.

In the discussion of Siderosis it was pointed out that the "spots" noted on the films of welders were due to iron oxide pigmentation and were not to be confused with silicosis. This observation is to be re-emphasized in view of the mention that silicon is often a part of the composition of welding rods. According to Tebbens and Drinker, chemical behavior of the $SiO_2$ determined in the fumes of the steel electrodes indicated that it was present in either a soluble silicate or an amorphous form of silica. No crystalline quartz is present in the fumes.

### Metallizing

"Metallizing" is a trade name which was applied to the metal spraying process a number of years ago. Because of its tremendous versatility, metallizing as a maintenance and as a production process finds far-flung applications. It can be used competitively in place of tinning, galvanizing, calorizing, electroplating, and welding under certain conditions.

The metallizing process as a whole involves three phases: (1) surface preparation, (2) application of the coating, and (3) finishing of the sprayed surface, if necessary.

All metallic, and in some cases nonmetallic, surfaces, require a preliminary preparation before the sprayed coating can be applied. The purpose of this preparation is to clean the surface thoroughly and to roughen it sufficiently so that the coating will adhere as it is sprayed on. There are several methods which can be used to accomplish this, but for commercial reasons there are only four which are practical.

On large flat areas or irregular surfaces a thorough sand or grit blasting will give the desired results, using a sharp abrasive only. On shafts or similar cylindric members preparation is accomplished by either rough threading or heavy blasting with coarse angular steel grit.

Either method of preparation roughens up the surface in such a manner that innumerable pores and crevices are created, into which the sprayed metal locks itself so securely that a splendid bond is achieved. Surfaces which are naturally porous, such as cement, plaster, wood, etc., require no preliminary preparation provided they are clean and free from paint, lacquer, or other sealers.

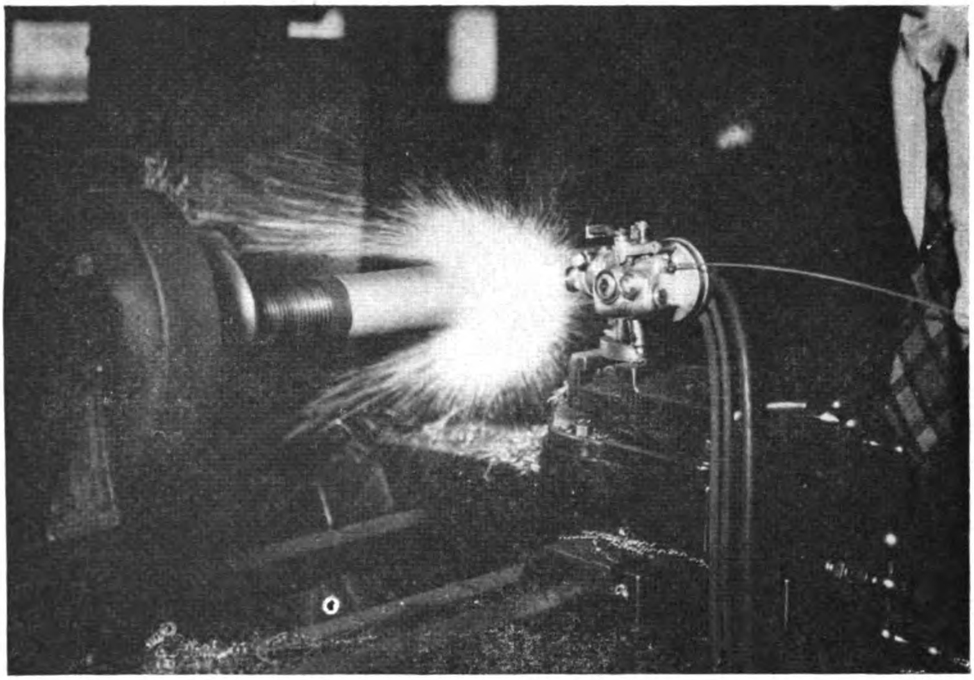

Fig. 95.—Metallizing a shackle pin. (Courtesy Metallizing Company of America.)

The next step in the metallizing process is the application of the coating by means of a metal spraying tool generally referred to as a metallizing "gun." This is a small, lightweight device consisting of a power unit and combustion unit united as an assembly. The power unit is made up of an aluminum housing which encloses a high-speed air turbine attached to a gear reduction, the latter running in a bath of fluid grease. Connected to the reduction gearing are the feed rolls which feed the wire or rod through the gun during the spraying operation. The combustion unit comprises the gas head, simultaneous control valve, wire nozzle, air cap, and turbine-regulating screw, the latter controlling the speed of the wire or rod as it passes through the nozzle (Fig. 95).

While the spraying unit is in operation the wire or rod is fed through the power unit at a uniform speed and on through the combustion unit into the apex of an oxyacetylene or oxypropane flame cone where it instantly is reduced to a molten state. As fast as the metal is melted, it is atomized by a concentric air blast which drives it at high velocity against the surface to be coated. As the minute particles of molten metal strike the prepared surface, they are driven into any crevice that may be in their line of flight, thus forming a strong, coherent coating of sprayed metal. Since metal spraying is a continuous operation, the coating can be built up to any predetermined thickness.

The last stage of the metallizing process is the finishing, and to what extent finishing will be necessary depends entirely on the purpose of the coating. Protective coatings that are sprayed on as a resistant against corrosion are rarely finished in any manner. Coatings that will be used for decorative purposes may be ground, polished, or given a chemical treatment to change their color. In finishing machine elements, it will depend on the nature of the metal sprayed as to whether the coating can be tooled or will require grinding. Practically all the metals except the higher carbon steels can be machined without trouble, but for a satisfactory finish on a high-carbon steel coating it will be necessary to grind, the hardness of this type of coating making it almost impossible to tool.

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# CHAPTER XXXVIII

## INDUSTRIAL HYGIENE — GENERAL

Industrial hygiene is popularly conceived as a survey by industrial hygienists and engineers who periodically go through a plant with queer-looking gadgets, sniffing and sampling the air in the hope of finding trouble. Some have defined industrial hygiene as the appraisal and control of industrial hazards. Actually, the ideal industrial hygiene program would concern itself with the optimum health and happiness of the worker *as well as* with his working environment.

An industrial hygiene program is effective only if it engages the combined efforts of the engineer, the hygienist, the chemist, *and the physician.* It should begin with an appraisal of the worker's physical and mental condition, by use of the pre-employment examination, and be followed by the physician's continuous control of the worker. In other words, the physician's appraisal and control of the worker is as important as the engineer's appraisal and control of the working environment. The physician's interest should extend beyond the four walls of the plant; it should include the factors which influence the worker's general health, his attitudes, his personal hygiene, his nutrition, and so forth.

Regarding the working environment, efficient control of hazards requires trained men and constant vigilance. These requirements are fulfilled to a very satisfactory degree in our larger industries but are rarely maintained in small plants. The owners of most small plants contend that they cannot afford a full-time hygienist or industrial engineer. Nor, if they could, are there enough trained specialists in this field to meet even the most urgent demand.

What, then, is the answer to the problem of small industry? First, these small plants should avail themselves of the consultation service offered by local or state agencies of the United States Department of Health, Division of Industrial Hygiene, or by private agencies capable of performing this service. But a consultation service is not the complete answer. At best it is only a periodic survey and usually limited to a specific problem. What every plant needs is a daily surveillance of its own housekeeping, a system whereby commonplace procedures maintain an orderly, clean, and healthy place in which men can work without incurring undue risk to their health.

The ordinary objectives of good plant hygiene can be attained in much the same manner as through the observance of the "safety first" program. In fact, it is merely the extension of such duties. For the plant as a whole or for the various departments, individuals chosen for their insight or background could be assigned the responsibility of maintaining good plant hygiene. This should never be a casual assignment. Those chosen should be given the opportunity to enroll in courses of industrial hygiene which are conducted in practically every city of any size in America. They should be given access to the literature such as is to be found in the National Safety News, in the publications of the Division of Hygiene of the United States Department of Health, or from their own state department of health.

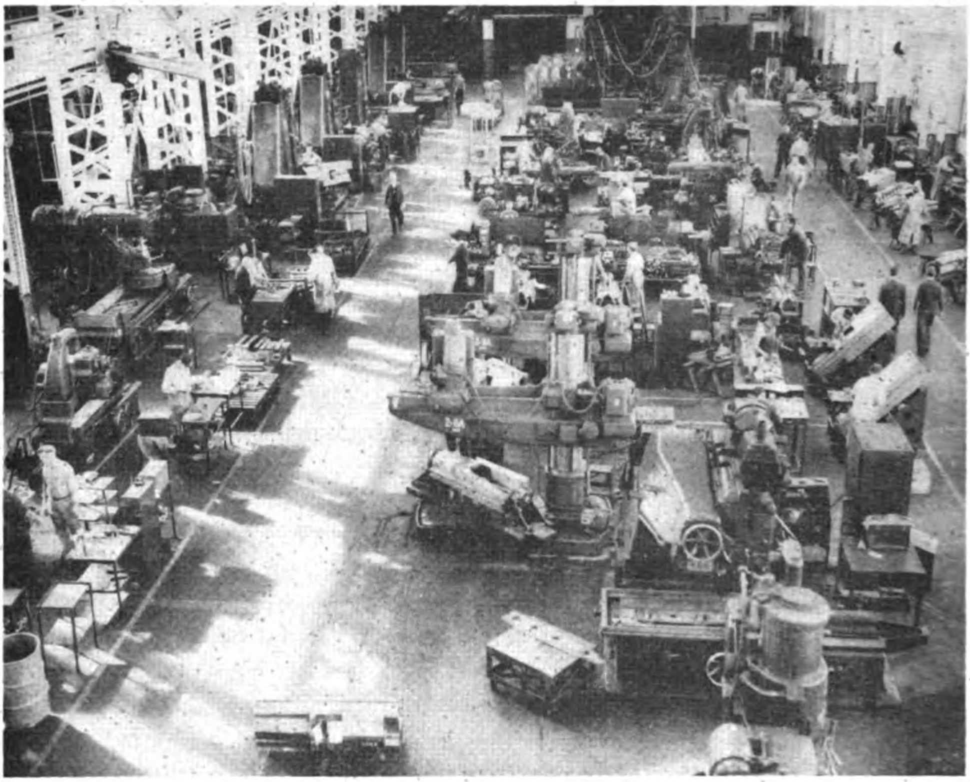

Fig. 96.—Example of good housekeeping. Note aisle space and arrangement. (Courtesy General Motors Corporation.)

**General Plant Hygiene.**—Good housekeeping is as essential to the welfare of the occupants of a plant as it is to the members of a household, nor do the methods differ to any great extent. In the home it is called dusting, sweeping, washing, heating, and airing the house; in the army it is called "policing up" the barracks or grounds; in the plant it is a part of industrial hygiene. In the safety records of any plant one rarely sees accidents charged to poor housekeeping, yet slovenly methods of storing or stacking stock, protruding objects, slippery floors, unkempt or crowded aisleways, and other instances of neglect and lack of imagination are all potential accident producers (Fig. 96).

Good general housekeeping can be achieved by extending the janitor service from the management's office into and throughout the plant proper. This should include not only sweeping and vacuum cleaning of the floors, but also of the work benches, the tools, and the machinery as well, especially those on which oil or grease is likely to accumulate. Spray booths should be scraped and cleaned each night and the dust should be removed from all areas where grinding, polishing, and such processes are being done.

Procedures which require only periodic attention should be scheduled and listed on a board rather than to be left to the memory of those responsible for such care. These include painting and checking of lighting, heating, air and water-purifying systems, fire hose, fire extinguishers, fire escapes, and safety and medical equipment such as stretchers, first-aid kits, oxygen tanks, and other emergency supplies.

Obviously, these various responsibilities should be divided among a number of individuals, but management should assign one person to whom all the others are accountable. Even though this person has had no previous training, he can, by application and study, become expert at his job and will, in addition, find it a most humane and interesting field of endeavor.

Before considering the control and appraisal of toxic hazards, a few plant and personal protection measures are herewith presented.

## Plant Lighting

Good lighting[1] is a fundamental factor in a safe, healthful, and agreeable working environment. It is also essential to the visual efficiency of the individual.

Estimates of the proportion of industrial accidents due to poor lighting have varied from 15 to 25 per cent. However, it is often difficult to point to the exact cause, and many injuries due directly or indirectly to poor lighting are attributed to other causes. Many instances of improved illumination have been followed by lowered accident rates.

**Natural Lighting.**—Advantage should be taken of available sources of daylight. Overhead diagonal sources are preferable to side windows alone, which are frequently sources of glare. Sawtooth, monitor, or skylight windows take full advantage of natural light.

These windows may be fitted with refracting or diffusing glass which will alter the direction of light and improve its distribution, particularly to distant parts of the room. Translucent coatings for windows on the sunny sides of a building are also helpful in avoiding glare.

Reflection of daylight from sources outside a building should be utilized. Light colors for the faces of structures, walls of courts, and sawtooth roofs are helpful. For maximum efficiency these surfaces should be kept clean and sources of glare avoided.

Sudden transition from brightly lighted to dim areas is hazardous. The result is often a momentary blindness while the pupil of the eye adjusts itself to the dimmer light. Graduations of light at the approaches to areas of different intensity will avoid this trouble.

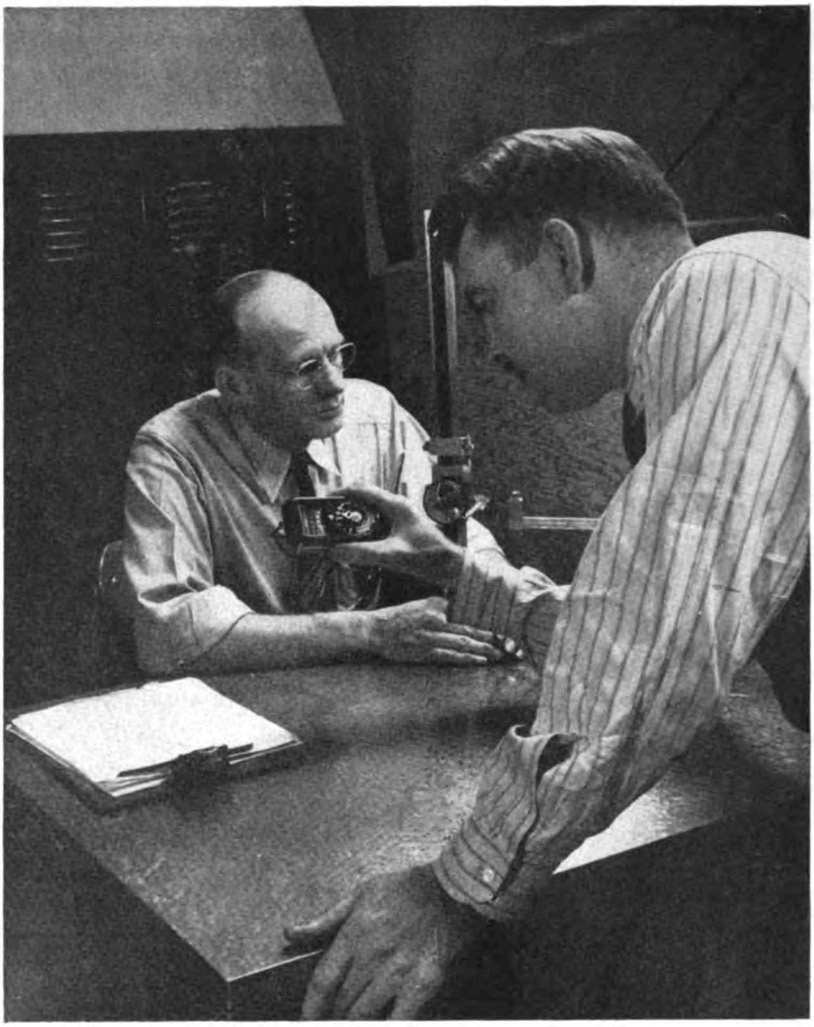

Fig. 97.—Checking to see if light is adequate.

**Artificial Lighting.**—Because natural light is undependable, especially in winter, artificial lighting has become the major source of industrial illumination for continuous operation; it is, of course, essential. Excluding overtime and night work, it will be found necessary to rely upon artificial lighting for 20 to 50 per cent of the total working hours.

Recommended Levels.—Standards of illumination for various types of work will be found in American Recommended Practice of Industrial

Lighting. The illumination levels are determined from readings of a light meter at the spot where light is needed (Fig. 97).

Footcandles for general overhead lighting range from 5 footcandles for storage areas to 50 footcandles where the seeing task is more exacting.

For many operations general lighting does not supply enough light at the point of operation for precision work. Supplementary lighting may be employed to provide 100 footcandles for exacting and prolonged seeing tasks such as fine bench and machine work. For extra fine inspection, as in making jewelry and precision instruments, 200 footcandles have been recommended.

Modern Light Sources.—The three common sources of artificial light are the incandescent lamp, the high intensity mercury vapor lamp, and the fluorescent lamp.

In spite of competition from newer types of lighting, the incandescent or filament lamp remains an important and versatile light source. It requires only a standard socket and the available current to provide light. The advantages are low cost, simplicity, and convenience, together with a pleasing quality of light.

The high intensity mercury vapor lamp has a relatively high efficiency but it requires auxiliary equipment, such as a transformer and current ballasting. The light consists mainly of violet, green, and yellow-green and is deficient in orange and red.

Mercury vapor lamps have low operating cost and long life but these advantages must be weighed against the less pleasing color of light, the relatively long restarting time, and the initial cost of equipment. These lamps are frequently used for interiors with high ceilings with alternate fixtures of mercury vapor and incandescent lamps.

Fluorescent lighting has grown steadily in popularity during the past six years. Fluorescent tubes are best suited for installation in large areas and they should be mounted high. There is still room for improvement in the fixtures designed to hold these tubes. Few installations at present are shielded and the bare tubes are too bright for eye comfort.

The advantages of fluorescent lighting are the high lumen per watt efficiency and their relatively low heat output, which is about one-fourth that radiated from filament lamps. They are therefore particularly useful in air-conditioned interiors.

Reflectors.—No lamp should be used without a suitable reflector. There are several fundamentals to be considered in choosing the types of units:

1. Desirable distribution of light and suitability for the interior involved
2. Sturdiness of construction

3. Efficiency of light output
4. Adaptability in case more light is desired
5. Economy of maintenance (cleaning and replacement)

GLARE.—Care must be taken to avoid glare. This has been described as "any brightness within the field ot vision of such character as to cause discomfort, annoyance, interference with vision, or eye fatigue." It may be direct or reflected. The former has more to do with visual discomfort because the sensitivity of the eye is disturbed, while the latter interferes with visibility by reducing the contrast between the reflecting object and the background. Reflected glare is often more irritating than direct glare for it is usually so close to the line of vision that the eye cannot avoid it.

If direct general lighting fixtures are mounted above the normal line of vision, glare will be greatly reduced. When supplementary lighting fixtures are installed, care must be taken that this source of illumination does not annoy either the workman for whom it is installed or others in the line of vision with it.

SHADOW.—Shadow is another defect of illumination which is an important cause of industrial accidents. It may annoy in two ways: by resulting in undesirable brightness contrasts and by interfering with visibility at the work place.

MAINTENANCE.—Efficient lighting is impossible without a systematic program of maintenance. Equipment may meet all requirements when installed, but dust and grime immediately start taking their toll of the light output.

The recommended method of establishing a maintenance schedule is to check the illumination periodically with a light meter. When illumination has decreased to 75 per cent of its original value, the lighting equipment should be washed with warm water and a detergent containing no free alkali.

To insure continued adequate illumination, even under favorable circumstances, it is necessary to plan the system to give initially at least 25 per cent more light than the required minimum.

The best lighting system, however, may be deficient if proper "color painting" is not maintained. Interior finishes serve as aids for good lighting and better housekeeping. White and light tints on walls and ceilings increase the diffusion of light and distribute it more evenly. Glare is minimized by the removal of dark backgrounds which contrast harshly with bright areas.

Industry has learned that there is no "all-purpose" paint. So many different needs and exposures are involved that it has been necessary to develop many types of pigments and vehicles to meet the many different and exacting conditions.

For plant maintenance there is available an amazing variety of finishes for wood, metal, or concrete. Actually, each finish consists primarily of a solid pigment, a liquid vehicle, a drier, and a volatile thinner, but each of these ingredients may vary considerably in its characteristics.

Industry has long recognized color as a valuable aid in the prevention of accidents as a means of identifying and calling attention to hazards. It has also been demonstrated that the use of color has a profound psychologic effect—boosting workers' morale by reducing monotony, fatigue, and loafing at the job. Cheerful surroundings are a strong antidote for absenteeism.

Color is a maker of moods. It can make people feel warm or cold, excited or tranquil, comfortable or depressed.

**Functional Use of Color.**—As an outgrowth of war needs, the American Standards Association's war committee prepared the "American War Standards Safety Color Code for Marking Physical Hazards and the Identification of Certain Equipment."

Briefly, the standard is a codification of certain widely recognized concepts regarding use of color for safety purposes.

Red is indicated (1) for the identification of fire protection equipment and apparatus, (2) for danger, (3) for "stop" signs.

Yellow is indicated for caution and for marking physical hazards such as striking against, stumbling, falling, tripping, etc.

Green is indicated for safety and for location of first aid equipment.

White, black, or a combination of the two, is indicated for housekeeping and traffic marking.

Much of the standard deals with suggested applications for these colors, such as the markings of safety cans, fire sirens, posts, handrails, unguarded edges of platforms, location of gas masks, etc. The code frequently refers to standards already approved.

The use of color markings is not intended to become a substitute for adequate guards or other safety measures but to supplement them. In fact, a warning is written into the standards that the "marking of a physical hazard by a standard color warning should never be accepted as a substitute for the complete elimination of a hazard wherever this is possible."

The increased use of color in plant housekeeping has called for a corresponding increase in the use of paints of all varieties.

Under a variety of descriptive names, such as "three-dimensional seeing," "color dynamics," and "optonics," paint manufacturers have developed scientific color schemes for machinery as well as for the walls and ceilings of industrial plants. The correct combination of color and light has proved its value in bettering industrial seeing conditions.

It calls for painting machinery in colors that reflect light instead of absorbing it. Thus, better seeing conditions are provided because the usable light at the immediate working point is increased. When the bodies of machines are painted in one color, and the working areas a contrasting hue, these danger points are "spotlighted" clearly.

### Explosion Hazards of Dusts, Gases, and Vapors

Explosive substances[2] commonly encountered in industry may be divided into the following three general classes:

1. Commercial explosives or chemical explosives, such as dynamite and gunpowder, which contain the oxygen necessary for rapid combustion.
2. Gases, vapors, and flammable liquids. These are explosive when mixed in certain proportions with air. (Flammable liquids are not, properly speaking, explosive; they are included because they give off explosive vapors.)
3. Explosive dusts. Practically all finely divided organic dusts, and many metallic dusts, are explosive when suspended in the proper proportion of air. As a general rule, the finer the dust, the greater the violence of the explosion.

**Gases, Vapors, Liquids.**—So many new developments are being made in manufacturing processes that it is impossible to give a complete list of gases, vapors, and liquids that might cause an explosion under favorable conditions.

It is difficult to classify many substances as either flammable or explosive. Under certain conditions some may not even burn; if conditions are just right they may burn slowly; under other conditions, an explosion might occur.

The explosion range of gases is given in Table XXI.

TABLE XXI

| SUBSTANCE | FLAME IGNITION PER CENT GAS TO AIR |
|---|---|
| Acetylene | 2.6—82 |
| Alcohol vapor | 3.5—19 |
| Benzine vapor | 3—4.8 |
| Benzol vapor | 3—6 |
| Carbon disulfide | 1—50 |
| Carbon monoxide | 12.5—74 |
| Coal gas | 8—23 |
| Ether vapor | 0—45 |
| Ethlyene | 3—34 |
| Hydrogen | 4.1—75 |
| Methane | 5—16 |
| Pentane | 1.3—7 |
| Toluol | 1—6 |
| Water gas | 12—67 |

The following lists give a number of the more common substances whose storage and use is attended by explosion hazards.

The explosive gases are listed here alphabetically, not in order of hazard. For explosion range of gases, see Table XXI.

Explosive gases

- Acetylene
- Carbon monoxide
- Ethylene
- Hydrogen
- Illuminating gas
- Natural gas
- Miscellaneous commerical gases

Flammable Liquids

Class 1 (Liquids in this class have a flash point below 25° F., closed cup tester)

- Ether
- Carbon bisulfide
- Gasoline
- Naphtha
- Benzol
- Collodion
- Liquefied petroleum gas
- Acetone

Class 2 (Liquids in this class have a flash point above 25° F. and below 70° F., closed cup tester)

- Alcohol
- Amyl acetate
- Toluol
- Ethyl acetate
- Methyl acetate

Class 3 (Liquids in this class have a flash point above 70° F. and below 187° F., closed cup tester)

- Kerosene
- Amyl alcohol
- Turpentine
- Fuel oil

Other flammable liquids in the following list should be included in the preceding classifications according to their flash points:

- Paints
- Varnishes
- Polishing solutions
- Dryers
- Cleaning solutions
- Illuminating oils
- Miscellaneous petroleum products

Safety cans with self-closing spouts are desirable for any liquid that will burn and compulsory for those in Class 1.

Red is the prescribed color for containers, which should be plainly marked to identify the contents.

Dust storage should be separate and as far removed as practicable from buildings in which it is collected.

Dust should not be stored in large quantities for any length of time on account of the danger of spontaneous ignition and increased damage from fire and explosion.

Storage bins and tanks should be small, of incombustible material, and vented above the roof.

## Respiratory Protection

Dust, fumes, and gases[3] are inevitable in many industrial processes. Some of these are toxic; others are of the nuisance variety. But all are objectionable.

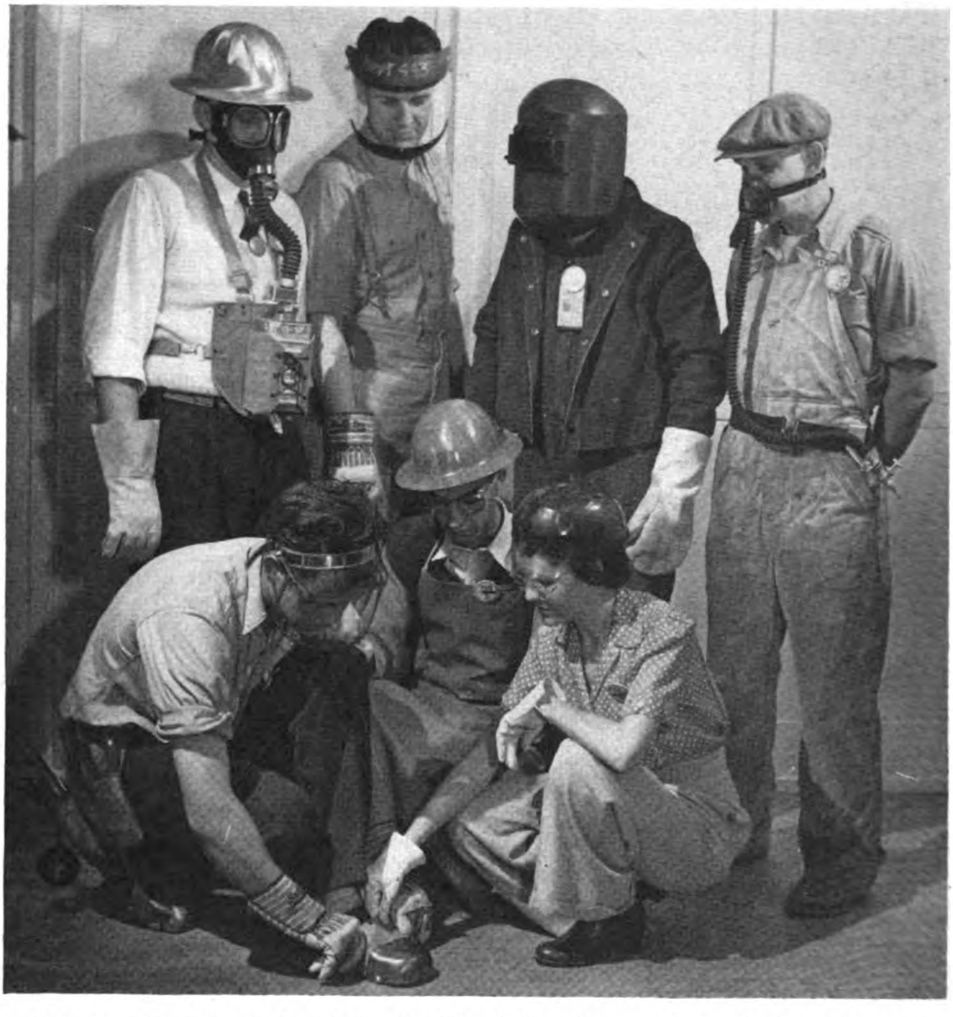

Fig. 98.—Shows the various types of safety devices given employees for both external and internal protection. Note safety shoes, helmet, face protection, welder's hood, airline mask, and canister mask. (Courtesy Douglas Aircraft.)

In many workplaces adequate ventilation has not yet been found practicable, and there are occupations where workers are exposed only to temporary concentrations of air contaminants. In such circumstances, respiratory protective equipment is needed.

Each type of equipment has a definite field of usefulness, but its limitations must be kept in mind. Reliable manufacturers and dealers therefore want to know the type of exposure before delivering equipment.

There for four general types of respiratory equipment:

1. Canister gas masks and chemical cartridge type respirators
2. Filter respirators
3. Supplied-air equipment
4. Self-contained oxygen-breathing apparatus (Fig. 98).

**Gas Masks and Cartridge Respirators.**—The gas mask and the cartridge type chemical filter respirator are useful for proection against solvent vapors and injurious gases. These two types of equipment are essentially the same except that the canister of the gas mask is considerably larger, contains a great deal more absorbent, and is consequently effective against higher concentrations and for longer periods of time than the chemical cartridge respirator.

The cartridge type respirator should be used only where the concentration of injurious material is low.

Canisters of gas masks are painted with identifying colors as follows:

1. Black—Organic vapors
2. White—Acid gases
3. Yellow—Organic vapors and acid gases
4. Green—Ammonia
5. Brown—Organic vapors, acid gases, and ammonia
6. Red—All industrial gases including monoxide, smoke, and fumes
7. White with green stripes—Hydrocyanic acid gas
8. White with yellow stripes—Chlorine

These canisters are approved by the United States Bureau of Mines for concentrations up to 2 per cent of most gases and vapors. If there is any doubt about the type of hazard or the gas mask being used, the manufacturer should be consulted.

Standards for chemical cartridge respirators were established during the past year by the United States Bureau of Mines. Schedules for approval of these respirators are now available. Approvals are granted only for protection against solvent vapors or vapors and particulate matter.

A gas mask is primarily emergency equipment, used under conditions of strain and excitement. Therefore, those who will have to use it should be trained carefully. Regular inspection is also essential to avoid deterioration of equipment when not in service.

The following items of training and maintenance are important:

1. Train each person in the proper method of putting the mask on and rapidly adjusting it to the face.
2. Have each person who may use the mask wear it long enough to become accustomed to the breathing resistance and to putting it on and taking it off.
3. Repeat training at regular intervals.
4. Set up a card for each mask to indicate the date of the latest inspection and replacement of the canister and the amount of use, if any, which the canister has had.
5. Replace canisters at least annually, whether or nor they have been used.
6. When the canister is replaced, carefully examine the facepiece, harness, hose, and headbands for leaks or deterioration; replace any defective parts.
7. Canisters are ordinarily supplied with seals to prevent the entrance of air until they are put into service. Remove these seals when the canister is installed.
8. Store the mask in a place readily accessible to the hazardous area, but one as cool and dry as possible.
9. If the mask is used for emergencies only, it is wise to replace the canister after each use.

**Filter Respirators.**—A mechanical filter respirator can be designed to give satisfactory protection against any form of particulate matter. The major items to be considered in the design are the resistance to breathing offered by the filtering element, the adaptation of the facepiece to various shapes of face, and the fineness of the particles to be filtered out. These particles determine the fineness necessary in the filter.

The American Standards Association Code Z-2 requires that the complete respirator show a resistance in excess of 50 mm. of water to inhalation at a rate of 85 liters of air per minute. The resistance on exhalation under the same conditions may not exceed 25 mm. Most good commerical respirators are actually held to considerably lower resistances.

In practice, respirators are approved only for use in the presence of nontoxic dusts, including silica and other pneumoconiosis-producing dusts, and for use in the presence of toxic dusts, fumes, and mists, in which case they must pass all these tests. In addition, the respirator must be capable of fitting various types of facial contours without leaking.

The industrial safety man should familiarize himself with the hazard for which a respirator is approved and should not permit its use for protection against hazards for which it is not designed. Mechanical filter respirators are of no value against solvent vapors, injurious gases, or lack of oxygen.

**Care of Equipment.**—When not in use, these devices should be stored in a clean, cool, dry place to retard deterioration of rubber parts. All parts, with the exception of canisters and cartridges, should be cleaned after each use. Facepieces and air lines or hoses may be washed with soap and water, rinsed in clean water, and dried. Filters and cartridges are replaced and the used ones discarded.

**Respirators With Impervious Clothing.**—The most extreme condition requiring respiratory equipment is in rescue work or emergency repair work that must be done in an atmosphere which is extremely corrosive to the skin and mucous membranes and is also acutely poisonous, or immediately hazardous to life. For these conditions a complete suit of impervious clothing, plus breathing equipment, must be provided. Complete suits of rubber or synthetic rubber with helmets and attachments for hose lines are available.

The material should be specified with sufficient mechanical strength to provide for rough handling and considerable abuse without tearing. The hose line connection should be made to the suit itself, as well as to the helmet, since the wearing of such an impervious suit for a long period is not only fatiguing, but also actually dangerous unless it is well ventilated.

**Hose Masks.**—The general rule for all respiratory equipment is that hose lines (that is, at least a one-inch hose connection), rather than air lines with connection to the compressed air system, are to be used in atmospheres immediately hazardous to life. The primary reason for using hose lines is that in case of failure of the air supply it is possible to breathe through a considerable length of hose.

Hose masks are not approved with more than 150 feet of hose or where inhalation resistance exceeds 2.5 inches of water, or the exhalation resistance exceeds 1 inch of water. Apparatus approved by the United States Bureau of Mines will meet these specifications. If additional hose is attached to existing hose masks, it should not exceed the prescribed length and should be approved for use with that type of mask. It should be connected by means of approved couplings.

The hose mask should always be used for any work which involves entering tanks or pits where there may be a dangerous concentration of dust, mist, vapor, or gas or an inadequate amount of oxygen.

Oxygen deficiency should be suspected in any iron or steel tank unless a test has shown that the supply is adequate to support life. The rusting of walls of an otherwise clean tank may deplete the oxygen supply dangerously. An inadequate amount of oxygen and the presence of dangerous gases should also be suspected in any tank which has held organic materials, such as grain or grain products, any type of oil, or any food material.

The hose mask should bear the approval of the United States Bureau of Mines, shown by an approval number printed upon the container, molded into the facepiece and molded or branded into other parts of the equipment.

Upkeep consists of oiling and mechanical attention to the blowers, inspection and periodic replacement of the inhalation and exhalation valves, and regular cleaning of the rubber parts.

Care should be taken to adjust the facepiece in accordance with the directions of the manufacturer. The blower is designed to furnish an excess of air sufficient to keep the facepiece under a slight positive pressure, but in case of blower failure, air must be drawn through the hose by the inhalation, and the facepiece and exhalation valve must withstand the resulting negative pressure without leakage.

The body harness needed to pull the hose lines requires inspection prior to each use. The minimum requirement is that the component parts of the harness shall withstand a pull of at least 250 pounds. No parts which are to be used subsequently should be tested by pulling, but they should be examined for signs of wear or deterioration. Any condition which appreciably reduced the mechanical strength should indicate replacement.

Where hose masks are supplied as emergency and rescue equipment, frequent and thorough training in their use should be given to those who may use them.

**Abrasive Blasting Protectors.**—In abrasive blasting, the requirements are the same as those for a hose mask or air-line respirator, with the addition that mechanical protection from abrasive particles is needed for the head and neck. This protection can be supplied either by an impregnated cloth hood or by a helmet of some rigid material. It should be covered both inside and outside with a plastic material, such as soft rubber, to increase both the comfort of the wearer and its resistance to the abrasive.

A window of glass protected from the abrasive by a 30- to 40-mesh fine wire screen should be provided. This window should be of laminated safety glass to prevent shattering in case of a heavy blow. It should also be free from color and from defects. Both the window and the protective screen should be readily replaceable.

**Oxygen Breathing Apparatus.**—When it is necessary to work in atmospheres immediately hazardous to life at distances from the source

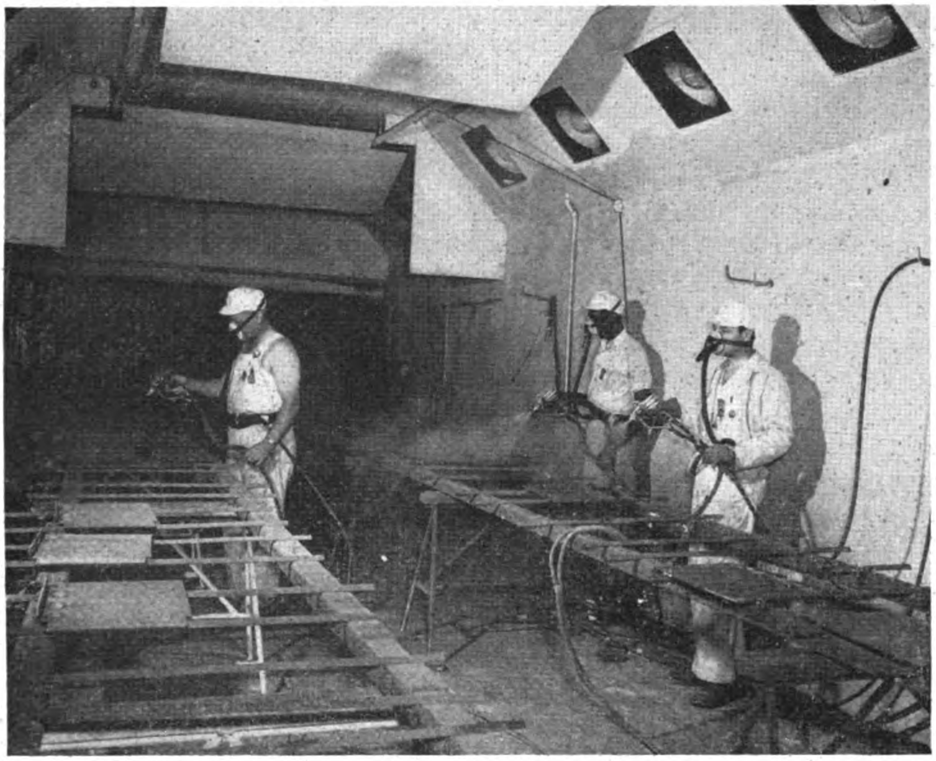

Fig. 99.—Spray paint booth, where the workers may be subjected to the fumes caused by other painters. They are, therefore, supplied with an air-line respirator. The booth also has a water wash.

of fresh air greater than the 150 feet provided by a hose mask, self-contained oxygen breathing apparatus may be used. In an atmosphere which is dangerously irritant or corrosive to the skin, it must be supplemented by impervious clothing. All such apparatus is somewhat difficult to handle and should be used only by specially trained persons.

Most oxygen breathing apparatus carries its oxygen in cylinders. A new apparatus has a small canister containing a chemical. Moisture from the breath starts a chemical reaction which liberates oxygen.

**Air-Line Respirators.**—For most industrial purposes, the air-line respirator for connection to compressed air lines provides essentially the same protection given by hose masks. It differs from the hose mask mainly in having hand-operated, quickly detachable coupling connected to the belt or body harness so that the operator can connect onto a compressed air hose, and also in containing a flow-limiting device with capacity to permit air flows between only 2 and 20 cubic feet per minute (Fig. 99).

Although not a requirement, it is extremely desirable that a trap and filter be installed in the compressed air line ahead of the masks to separate oil, water, scale, or other extraneous matter from the air stream. An air-pressure regulator in the line is required if the air is supplied at a pressure in excess of 25 pounds per square inch and, in addition, a pressure release valve which will operate if the regulator fails.

The air-line respirator furnishes complete protection against any atmosphere not immediately dangerous to life and is the most desirable protection for industrial operations requiring the continuous use of a respirator. Even where other types of respirators give adequate protection, they offer a breathing resistance not present in the supplied air type and are consequently much more fatiguing to wear. Its limitations, however, should be considered in the installation.

The air supplied must be cleaned of oil mist, rust, or other dust and must be free of carbon monoxide or other gaseous contaminants. To obtain clean air, the compressor intake must be kept away from all sources of contamination. The compressor should be well maintained and must not run too hot, as dangerous amounts of carbon monoxide can be produced by decomposition of lubricating oil.

The air, as delivered to the mask, should be of a comfortable temperature and humidity. The work of compression and re-expansion is often sufficient to produce a temperature and humidity widely different from that of the air entering the compressor. Provision should be made for automatically conditioning that part of the supply going to the respirator with respect to temperature and water content as well as with respect to pressure.

**Cleaning and Maintenance of Respiratory Devices.**—These devices should be sterilized before being passed from employee to employee. They should be capable of withstanding sterilization by immersion for not less than ten minutes in 40 per cent formaldehyde solution diluted with nine times its own volume of water, by boiling for at least five minutes, or by exposure for not less than ten minutes to a moist antiseptic vapor such as the vapor of formaldehyde.

Quarternary ammonium salt disinfectants can be particularly recommended, since they are not especially irritating if rinsing is complete after sterilization.

It is generally not possible to sterilize headbands, at least not the elastic bands, but they may be washed with soap and water and replaced when the respirator is transferred to another user.

Where a number of respirators are in regular use, a central station should be set up for care and maintenance, as well as for the care and maintenance of other items of personal protective equipment.

Each employee should be provided with two respirators and either a locker or a hook at the central station. The respirators should be branded or tagged with a number to indicate to whom they are assigned.

Whatever method of identification is used, it should be permanent enough so that the marking cannot be changed inadvertently and without a certain amount of effort.

The respirator should be turned into the central station at the end of each shift to be cleaned and repaired about once a week when retained by the same employee.

Where the maintenance crew works several shifts, one respirator per employee may be sufficient, but, in general, it will be necessary to have one complete unit in the process of cleaning and repair while the other unit is being worn.

## Eye and Face Protection

The eyes and face are exposed to injury in many operations from flying objects, splashes of molten metal or corrosive liquids, and harmful rays.[4] Because of the high percentage of permanent disability in eye injuries, this phase of protection has a prominent place in any safety program.

Equipment for the protection of the eyes and face include goggles of various types, face shields, acid hoods, welding masks and helmets, and babbitting masks.

**Impact Goggles.**—The standard eye protection against flying objects is the spectacle type or the eye cup goggle. Specification of the particular type to be worn will depend upon the nature of the job and the eye hazards involved.

Spectacle goggles are suitable for light or moderately heavy work, such as grinding, machine work, and assembling where working positions are not too close. Side screens of metal or transparent plastic are desirable to provide protection against light objects flying from the side. They should be used where operations are close together, or where more than one employee works on the same operation (Fig. 100).

The frames must be rigid enough to hold the lenses in proper position in front of the eyes. The nose bridge should be adjustable, or goggles should be available in enough sizes to fit various shapes of faces. Frames should be of corrosion-resistant material which will not irritate or discolor the skin. Goggles should be designed for easy cleaning and sterilization.

Cup goggles are designed for heavy duty. They are used for heavy grinding, machining, chipping, riveting, work with molten metals, and similar operations.

The cup should be wide enough to protect the eye socket and distribute the impact from any blow over a wide area of the facial bones. The cup should be flame-proof, corrosion resistant, and not irritating to the skin. To give the widest possible field of vision, goggles should be fitted as close to the eyes as possible without bringing the eyelashes into contact with the lenses and the lenses should be comparatively large. The minimum permissible size for oval lenses is 48 mm. in the vertical direction and 44.5 mm. in the horizontal. Round lenses should be 50 mm. in diameter. Lenses should have no appreciable distortion or prism effect.

To meet Federal strength specifications, a lens must stand an impact of a 22 mm. (approximately 44 grams) steel ball dropped from a height of 127 cm., both when supported in the eye cup of a goggle and when supported on a rubber gasket on a wooden tube. Lenses broken by impact should show only radial cracks. Concentric cracks around the point of impact, or scalding of the glass, are considered indication of improper heat treating.

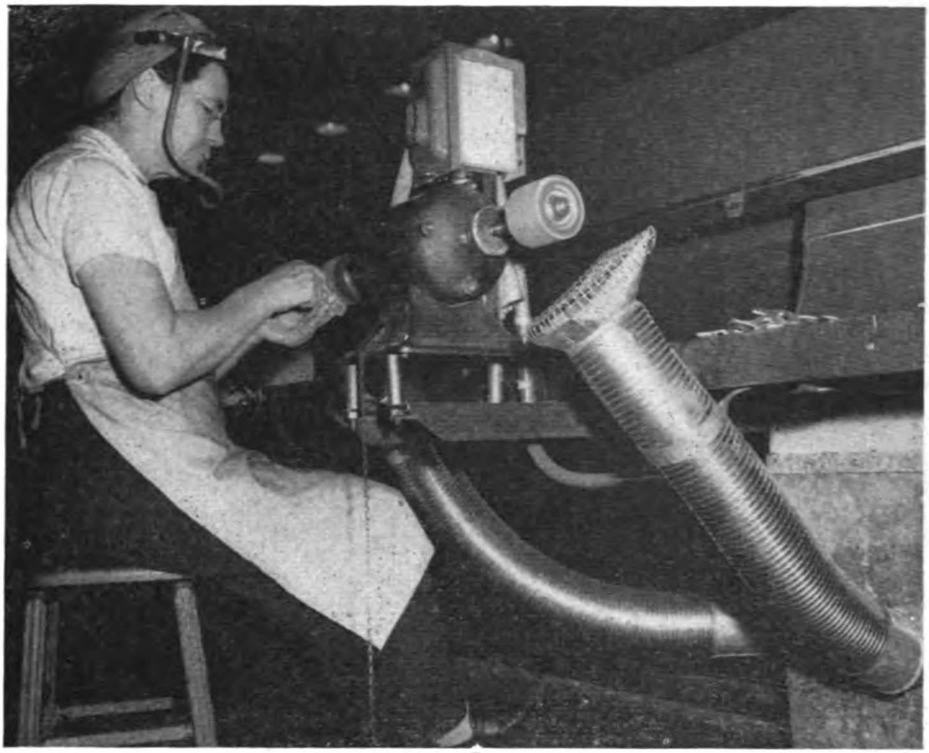

Fig. 100.—Dust-collecting units for burring and filing of aluminum castings. Worker also wears face protection.

**Corrective Goggles.**—When corrective lenses are required, the correction is preferably ground into the protective lens. Cover goggles may be worn over ordinary spectacles but this requires a cup deep enough to admit the whole spectacle. The deep cup noticeably restricts the field of vision.

Where the correction is critical, the cover goggle may also displace the spectacle enough to introduce serious distortion of images with resulting eye strain. Hardened corrective lenses may be installed in spectacle type goggles with side shields.

A fundamental requirement of any type of goggle is that the lens shall be broken by a blow which is not suffiicient to drive the lens out of the frame.

To be worn comfortably, goggles must be properly fitted. The company furnishing the goggles will generally be willing to train one or more employees in the proper method of fitting, adjusting, and maintaining goggle frames. If this cannot be arranged, the fitting should be done by an optometrist or oculist.

**Face Shields.**—When the hazard is only from light impact, such as from sparks which fly from spot welding operations on sheet metal, enough protection may be provided by a plastic face shield. No standard specifications have yet been set up for face shields, but the headbands should be adjustable to provide a comfortable fit for all head sizes; there should be an easily operated joint to permit pushing the shield up from the front of the face.

The plastic sheet should be nonflammable, free from scratches or other flaws which will introduce distortion into the images, and heavy enough to resist distortion either by light impact or from varying conditions of temperature and humidity.

**Acid Hoods.**—Head and face protection from splashes may be provided in a variety of ways, depending upon the severity of the hazards. The most complete protection is given by a soft rubber or rubberized fabric hood with a glass or rigid plastic window.

No strength tests are specified for such hoods but, if they are rubber, they should at least pass the requirements for the rubber used in respirators. Tensile strength should be no less than 1,700 pounds per square inch, and there should be no loss of more than 20 per cent after seven days in air at 70° C.

Because they are extremely hot, particularly in warm, humid surroundings, these hoods may be supplied with an air line. When so supplied, the wearer should be equipped with a harness or belt exactly like that on an air-line respirator for the control of the supply line.

Where protection is required only from limited direct splashes, good protection is provided by an impervious hat and by a face mask of any material not affected by the liquid being used.

No approval tests have been specified for such masks. Obviously, they should be shaped to cover the whole face and be curved under the chin and over the forehead. They should be supported from the head, band or head harness so that they can be easily tipped back clear of the face. The head harness should be easily removed toward the front when the mask is wet with corrosive liquid.

**Chemical Goggles.**—"Splash-proof" eye cup goggles should be worn under the face mask. They should be worn when corrosive liquids are being handled without the use of a face mask. The goggles should be made of soft, flexible rubber and should be equipped with lenses at least 44.5 mm. in diameter. They may have baffled ventilation openings if the vapor of the liquid being handled is not irritating to the eyes.

If the goggles are of a nonventilated type for protection against vapor as well as against splashing, some nonfogging device should be provided, such as a little water inside for clearing the lenses, or a compound to be rubbed on the lens.

No strength tests are specified for such goggles since they are not intended to resist impact. However, they may be provided with heat-treated lenses with considerable advantage.

**Welding and Babbitting Masks.**—Somewhat specialized protection is required from the splashes produced in the pouring of molten metals and from the splashes and radiation produced by welding. There is a serious amount of radiation from various hot operations but it is ordinarily in the infrared and usually not harmful. In furnace operations, there is frequently need for protection from visible glare. "Glass blower's cataract" has been attributed to long-continued exposure to infrared radiations from molten glass.

Certain industrial operations require only a filter lens to cut down the intensity of the visible glare. For these operations, protective lenses might be chosen by their appearance. However, it is better to specify the color and the total reduction in radiant energy required.

**Harmful Rays.**—The shade numbers of the standard filter lenses in protective goggles and arc welders' shields are determined by the visible density of the glass as outlined in National Bureau of Standards Handbook H24. There is a further requirement for glasses of each density as to the maximum transmission of infrared and of ultraviolet at various wave lengths. Filter lenses should be chosen and specified according to these shade numbers and required to pass these transmission tests whenever the reduction of other than visible radiation is necessary.

For protection against visible glare, the spectacle type of goggle, with opaque side shields for the worst conditions, is adequate. For conditions where no metal may spatter and visible glare must be reduced, as in acetylene welding, the cup type of goggle should always be specified.

To protect against ultraviolet and infrared radiation, as well as against visible glare, the protective lenses should be installed in some type of face shield, either hand held or supported on a head harness. The shield should be made of a nonflammable material opaque to the dangerous radiation and a poor conductor of heat. A metal shield would not be desirable for such use because it would heat up under infrared radiation.

There are conditions, such as the pouring of low-melting metals, where protection against radiation is not necessary but where it is desirable to protect the head and face against splashes of the metal. A face shield, similar to an arc welder's face shield, but made of wire cloth which provides much better ventilation than a solid shield, can be used.

Good visual hygiene includes more than just mechanical protection of the eyes. It is management's obligation to determine the visual fitness of the applicant for employment and then attempt to maintain or improve upon the visual status of the applicant. Testing of the eyes in the pre-employment examination is a part of the placement program. This testing, however, must not be confined to the old, traditional measurement of monocular acuity at twenty feet with the Snellen chart. An applicant who can pass the Snellen test perfectly at twenty feet might be exceedingly inefficient if assigned to work at ten-inch distance.

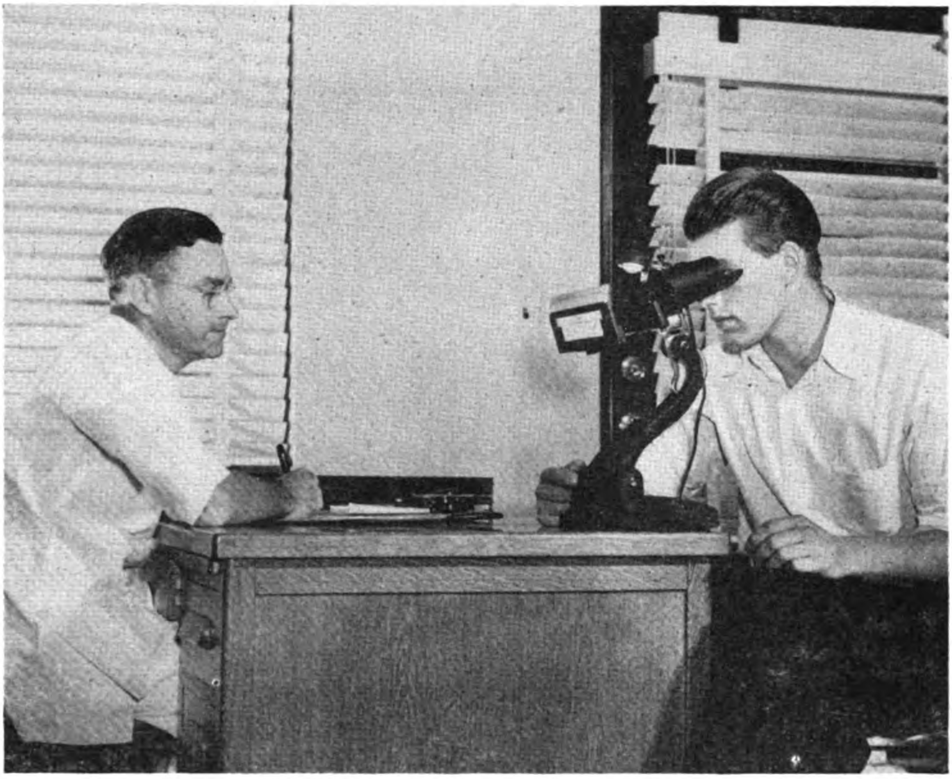

Fig. 101.—Eye examination.

Hedwig Kuhn has long been insisting that industry should demand of each employee an examination which will include a record of:

1. Acuity for distance and near vision without glasses

2. Acuity for distance and near vision with glasses
3. Stereopsis
4. Muscle balance for distance and near vision
5. Color appreciation level

This above type of examination is obviously designed to render men safe for jobs and jobs safe for men. As Kuhn points out, a crane operator with 20/15 O U with stereopsis may have a marked muscle imbalance and be doubly dangerous because his defect would go unrecognized under the traditional Snellen test. He "starts his day well, but fatigue breaks down his coordination as the day progresses and he ends his day (visually speaking) unstable and clumsy." Many other examples

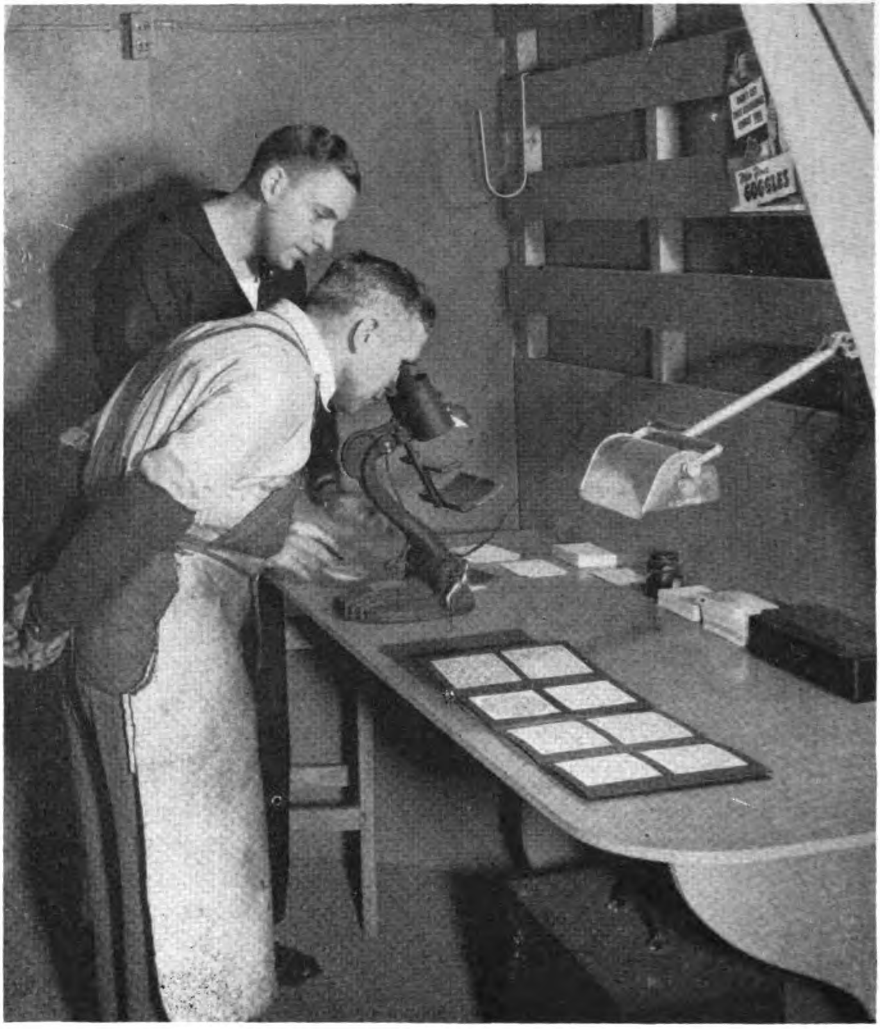

Fig. 102.—Interior view of portable visual survey trailer. (Courtesy U. S. Navy, Mare Island.)

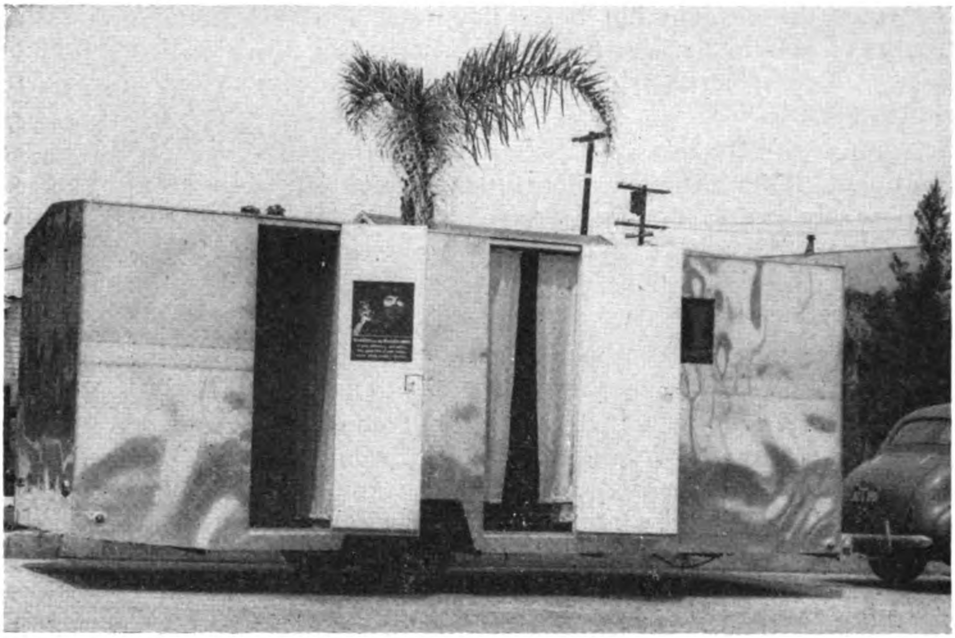

Fig. 103.—Visual survey trailer used in Los Angeles industries. This trailer is taken to the plant for eye examinations.

could be cited where men in specific jobs require specific vision. Those in charge of pre-employment and periodic physical examinations would do well to add to their library the textbook, *Industrial Ophthalmology* by Kuhn.[5] (Figs. 101 to 103).

### Preventing Skin Infections

Skin ailments are the most common of occupational diseases. In an average prewar year their cost to the nation was estimated at considerably more than $100,000,000 a year in medical care and compensation alone.[6]

New industries, particularly those which were concerned with the war effort, contributed many new causative agents and a greatly increased exposure. However, experience has shown that highly toxic or irritating materials are not the only causes of dermatitis.

While the high-powered irritants are serious offenders, lack of cleanliness has been established as a factor in a large proportion of dermatitis cases. Lack of lavatory facilities and proper cleansing agents, dirty work clothes, and careless washing habits all contribute to personal uncleanliness.

Since the cause is a factor in both the treatment of dermatitis and in the prevention of recurrences, every effort should be made to determine the causative agent. In doubtful cases the patch test is often employed.

Once the ailment has been diagnosed as occupational dermatitis, treatment should be directed toward protecting the skin from further injury. As a general principle, only mild medicaments are advised in acute cases.

Many with the less serious cases are permitted to work while under treatment. They often develop an immunity after recovery. Workers with severe cases should be removed from the irritant while under treatment. When a worker suffers recurring attacks, he should be transferred to a job where he does not come in contact with the offending substance.

**Skin Cleansers.**—Washrooms should be supplied with a good industrial skin cleaner. Such a cleanser should be freely soluble in hard, soft, hot, or cold water. It should not contain abrasive or irritant scrubbers nor added free alkali. Special cleansers should be provided for workers who have dermatitis or who cannot use the ordinary soaps without damaging the skin. Sulfonated castor oil, neutral in reaction, to which 2 per cent of a synthetic wetting agent is added, has been recommended for such workers.

**Cutting Oils.**—Cutting oils are a frequent cause of dermatitis wherever cutting and turning of metals is performed. The oils are divided into two main groups, soluble and insoluble.

The soluble cutting oils are less likely to cause dermatitis, although they usually contain bacteria. The insoluble oils are usually sterile, yet they are the principal causes of cutting oil boils and dermatitis. The metal slivers in them may wound the skin; the sulfur, chlorine, and inhibitors they contain may irritate the skin; the petroleum oil itself may plug the pores, causing comedones which may form boils if infected by bacteria from dirty clothing or unwashed skin.

Solvents, which are troublesome in many industries, are used in munitions plants for cleaning and degreasing shells, in spray gun stenciling, and in renovating old projectiles. Kerosene, petroleum spirits, chlorinated hydrocarbon compounds, Stoddard solvent, and alkaline solutions are among the solvents used. All can cause dermatitis by their defatting action on the skin. They may also cause allergic dermatitis.

**Safety Clothing.**—Garments made of impervious materials and protective applications for the skin are important measures in the control of occupational dermatitis.,

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2. National Safety News, March. 1946.
3. National Safety News, March. 1946.
4. National Safety News, March. 1946.
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# CHAPTER XXXIX

## INDUSTRIAL HYGIENE—GENERAL—Cont'd

### Control of Hazards

Industrial engineers have established the following fundamental methods for the purpose of minimizing and controlling industrial hazards:

1. Substitution
   Substances that are nontoxic or of low toxicity can usually be substituted for the more dangerous materials, as, for instance, shot blasting instead of sand blasting, or the use of a petroleum solvent in place of benzol.
2. Isolation
   Many processes can be enclosed so that the operators are not directly exposed to toxic substances. Also, dangerous processes should be carried out in isolated areas, away from workers not engaged in that specific process.
3. Exhausting
   Open windows, doors, fans, and ceiling or floor ducts may be sufficient for the removal of many nuisance dusts, vapors, or odors, but whenever the process involves toxic substances likely to concentrate in the breathing zone of the worker, such substances should be removed by "local exhausts;" suction, hoods, slots, and downdrafts for adequate ventilation are examples.
4. Wet process
   When dust cannot be properly exhausted, it can be controlled or minimized by wetting down the dust before and during operations. This should be done in many types of mining, drilling, sanding, buffing, etc.
5. Personal protection
   The various types of respirators and self-contained air equipment have been previously described, and their proper use has been indicated.

Of the various methods just mentioned, the one which needs to be discussed more freely is that of exhausting. The methods for exhausting toxic substances are being improved constantly. It is advisable for the industrial physician and plant engineer to subscribe to journals which periodically describe new methods and new designs. The following publications are recommended: *The Journal of Industrial Hygiene and Toxicology; Heating and Ventilation; Industrial and Chemical Engineer-*

*ing;* also the publications of the Industrial Hygiene Foundation of America, the Industrial Hygiene Research Laboratory, the National Institute of Health, Bethesda, Maryland, the Division of Industrial Hygiene, New York State Department of Labor, and the United States Public Healh Service.

**Manner and Method of Exhausting.**[1]—Hoods should be placed as close to the source of dust or fumes as possible, with due regard to the movements of the operator. When the hood must be placed at some distance above the machine, it should be built large enough to take care

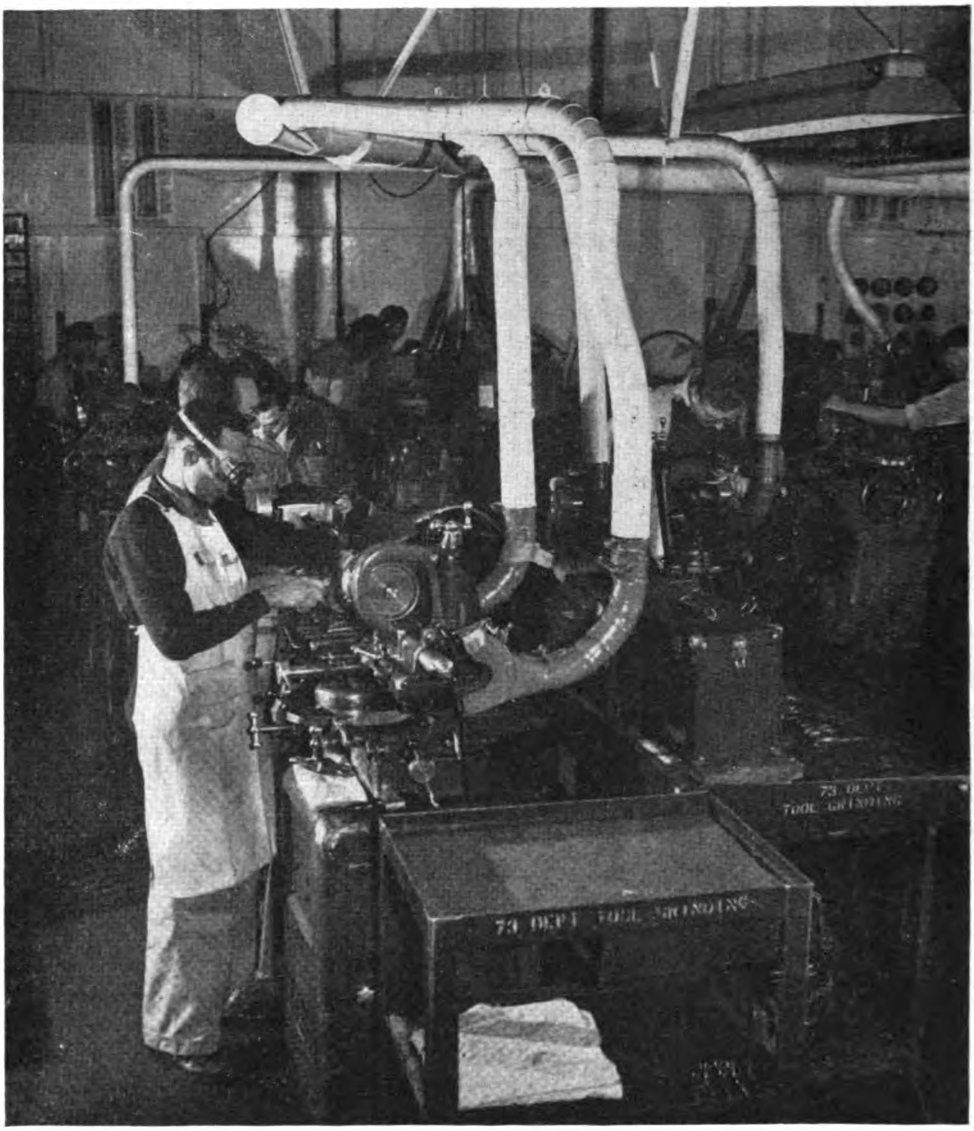


Fig. 104.—Tool grinding; dust drawn out into a central trap.

of the diffusion of the material which is usually quite rapid. For those substances which are lighter than air, the hood should be over or above the machine, and where a heavy vapor or dust-laden air at ordinary temperature is to be removed, horizontal or downflow type hoods should be used. The objective to keep in mind in all cases is to take advantage of the natural tendency of the material to move upward or downward.

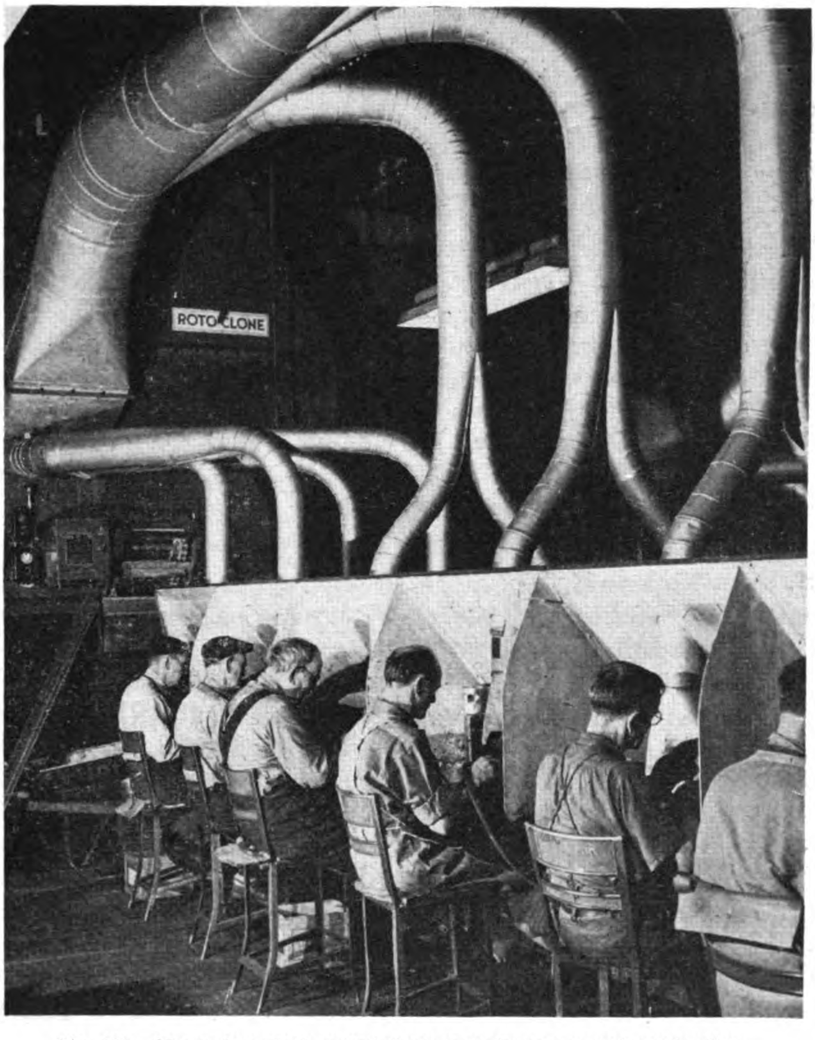


Fig. 105.—Magnesium dust pulled through water to prevent combustion.

The removal of dusts or fumes by means of exhaust hoods requires a movement of air at the point of origin sufficient to carry them to a collecting system. The air velocities necessary to accomplish this depend upon the physical properties of the material to be eliminated and the direction and speed with which it is thrown off. If the dust to be removed is already in motion, as is the case with high-speed grinding wheels, the hood should be installed in the path of the particles so that a minimum air volume may be used effectively. It is always desirable to design and locate a hood so that the volume of air necessary to produce results is as small as possible (Fig. 104; also Fig. 100).

The static suction at the throat of a hood is frequently used in practice as a measure of the effectiveness of control. This is of considerable value where exhaust systems adapted to particular operations have been standardized by practice. It must be remembered, however, that the suction is only a rough measure of the air volume handled and consequently of the air velocity at the opening of the hood. The elimination of any dusty condition requires added information concerning the shape, size, and location of the hood used with regard to the operation in question (Fig. 105).

In some states, grinding, polishing, and buffing wheels are subject to regulation by code. The static suction requirements, which range from 1½ to 5 inches water displacement in a U tube, should be followed, although in several instances they may appear to be very high. Frequently, in these operations, a large part of the wheel must be exposed and the dust-laden air within the hood is thrown outward by the centrifugal action of the wheel, thus counteracting useful inward draft. This tendency may be diminished by locating the connecting duct so as to create an air flow of not less than 200 feet per minute about the lower rim of the wheel.

**Hoods.**—No set rule can be given regarding the shape of a hood for a particular operation, but it is well to remember that its essential function is to create an adequate velociy distribution. The fact that the zone of greatest effectiveness does not extend laterally from the edges of the opening may frequently be utilized in estimating the size of hood required. Where complete enclosure of a dusty operation is contemplated, it is desirable to leave enough free space to equal the area of the connecting duct. Hoods for grinding, polishing, and buffing should fit closely but at the same time should provide an easy means for changing the wheels. It is advisable to design these hoods with a removable hopper at the base to capture the heavy dusts and articles dropped by the operator. Such provisions are of assistance in keeping the ducts clear. Air volumes used to control many dust discharges may often be reduced by effective baffling or partial enclosure of an operation. This procedure is strongly urged where dusts are directed beyond the zone of influence of the hood.

**Large Open Hoods.**—Large hoods, such as are used for electroplating and pickling tanks, should be subdivided so the area of the connecting duct is not less than one-fifteenth of the open area of the hood. Frequently it will be found necessary to branch the main duct in order to obtain a uniform distribution of flow. Canopy hoods should extend six inches laterally from the tank for every twelve-inch elevation, and wherever possible they should have side and rear aprons so as to prevent short-circuiting of air from spaces not directly over the vats or tanks. In most cases, hoods of this type take advantage of the naural tendency of the vapors to rise, and air velocities may be kept low. Cross drafts from open doors or windows disturb the rise of the vapors and therefore provision must be made for them. The air velocities required also depend upon the character of the vapors given off, cyanide fumes, for example, requiring an air velocity of approximately 75 feet per minute on the surface of the tank, and acid and steam vapors requiring velocities as low as 25 to 50 feet per minute.

**Lateral Exhaust System.**—The lateral exhaust method, as used for chromium-plating tanks, is often preferred to the canopy type hoods. This method draws air and fumes laterally across the top of vats or tanks into slotted ducts at the top and extends fully along one or more sides of the tanks. The slots are two inches wide and for effective ventilation a 2,000 feet per minute exhaust air velocity at the slot face is advisable. In addition, the duct should not be required to draw the air laterally for a distance of more than eighteen inches and the level of the solution should be kept six to eight inches below the top of the tanks (Fig. 106).

**Flexible Exhaust System.**—The flexible exhaust tube method may be advantageously used for removing dust or fumes. Flexible tubes having one end connected to an exhaust system and a slotted hood attached to the other end may be shaped at will to fit in with industrial processes without affecting the ease of operation. Efficient dust or fume removal may be had with use of relatively small exhaust volumes. This type of system may be used on swing grinders, portable grinding wheels, soldering operations, stone cutting, rock drilling, etc.

**Spray Booths.**—In the design of an efficient spray booth, it is essential to maintain an even distribution of air flow through the opening and about the object being sprayed. While in many instances spraying operations can be performed mechanically in wholly enclosed booths, the volatile vapors may reach injurious or explosive concentrations. At all times the concentrations of these vapors, and particularly those containing benzol, should be kept below 100 parts per million. Spray booth vapors are dangerous to the health of the worker and care should be taken to minimize exposure to them (Figs. 107 and 108).

The exhaust duct for a spray booth should be located in a horizontal position slightly below the object sprayed. Stagnant regions within the booth should be carefully avoided or should be provided with exhaust. The air volume should be sufficient to maintain a velocity of 150 to 200 feet per minute, over the open area of the booth, and the vapors may be discharged through a suitable stack to permit dilution, but it is better practice to pass the fumes or vapors through baffle type washers or scrubbers designed for efficient spray fume removal.

**Protection Against Corrosion.**—The removal of gases and fumes in many chemical plants requires that metals used in the construction of the exhaust system be resistant to chemical corrosion.

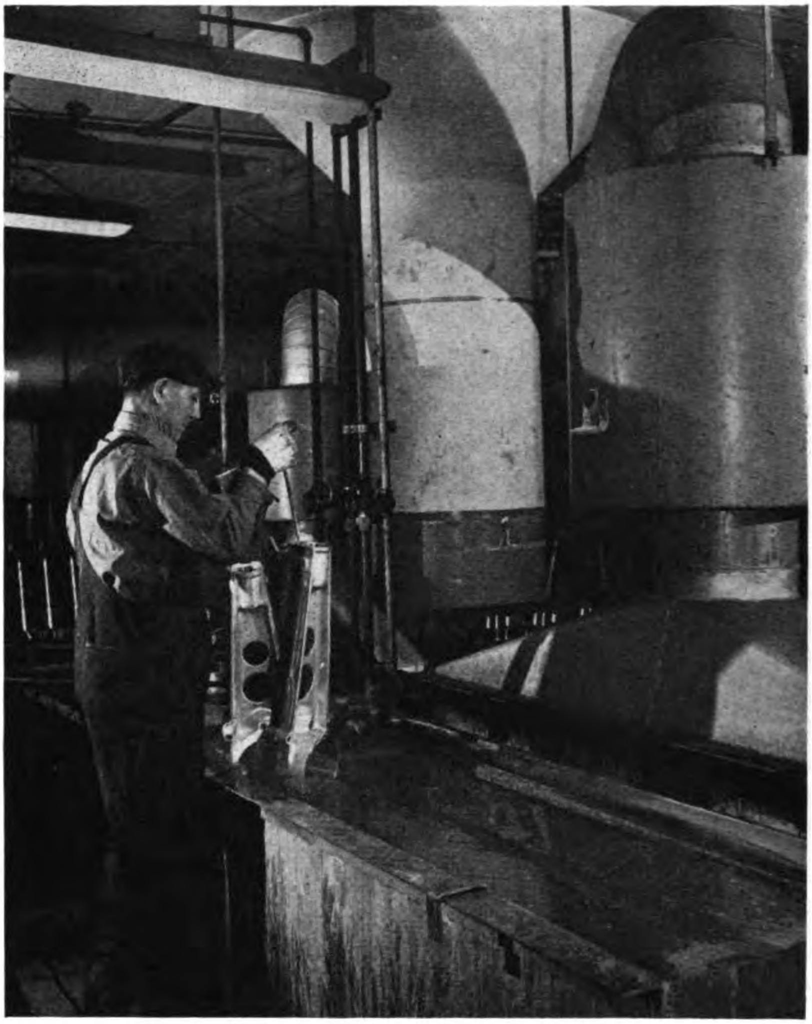


Fig. 106.—Acid fumes drawn off rinse bath. Note large lateral exhaust.

In an excellent series of four articles dealing with ventilation of solvents, Hemeon[2] recommends that the industrial design engineer should construct ventilation and exhaust systems which are directed not only to the prevention of poisoning but to the maintenance of an atmosphere that will be tolerable and even comfortable to all workers: the ventilation design should be satisfactory from the standpoint of smell and safe by toxicologic standards in the presence of vapors which are not unpleasant, even in dangerous concentrations. In short, Hemeon disagrees with the contention that engineering designs should only attempt to keep the concentration of toxic substances below the maximum permissible concentrations established by industrial hygienists.

**Application and Design of Duct Exhaust Systems.**[3]—Where the plant layout will permit, the fan and dust collector should be located at a central point, with main and branch ducts radiating to the various sections served. Equipment to be exhausted should be so arranged that, in so far as possible, short or large diameter branches are at the extremities of the duct system, while long or small diameter branches are near the exhaust fan.

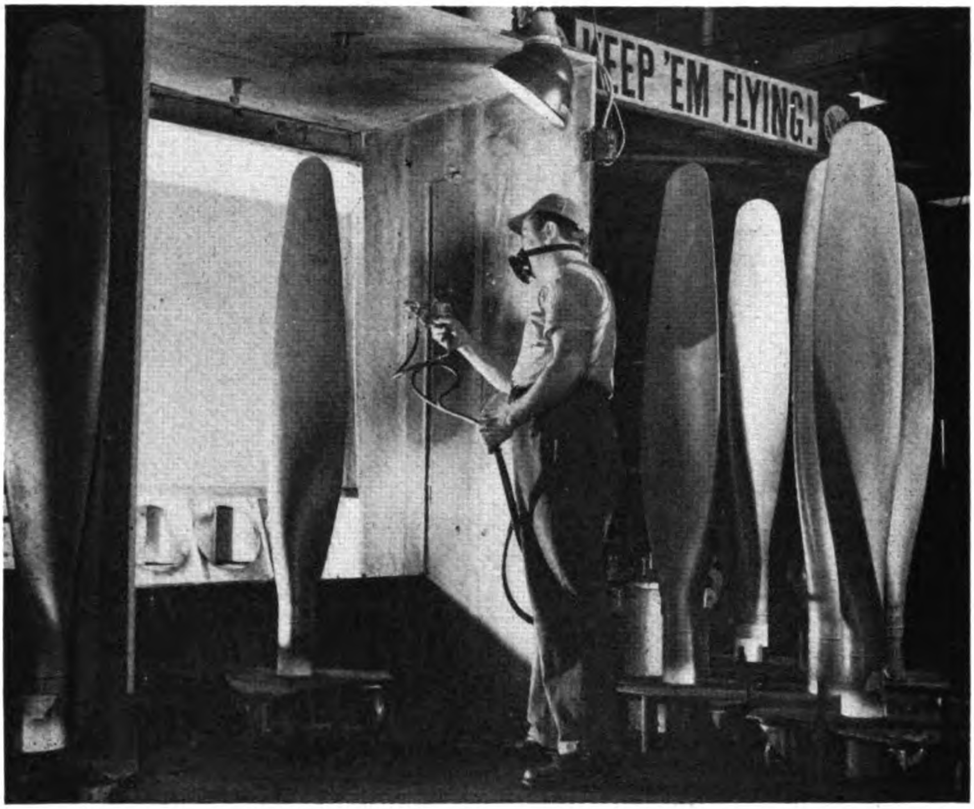

KEEP 'EM FLYING!

Fig. 107.—Spray paint booth. Running water collects excess paint while air ducts draw off fumes. Note individual respirator protection.

With scattered processes it is often advisable to separate the equipment to be cared for into several compact sections and to provide a separate exhaust fan and duct system for each section. This arrangement of individual systems usually reduces the full load power consumption

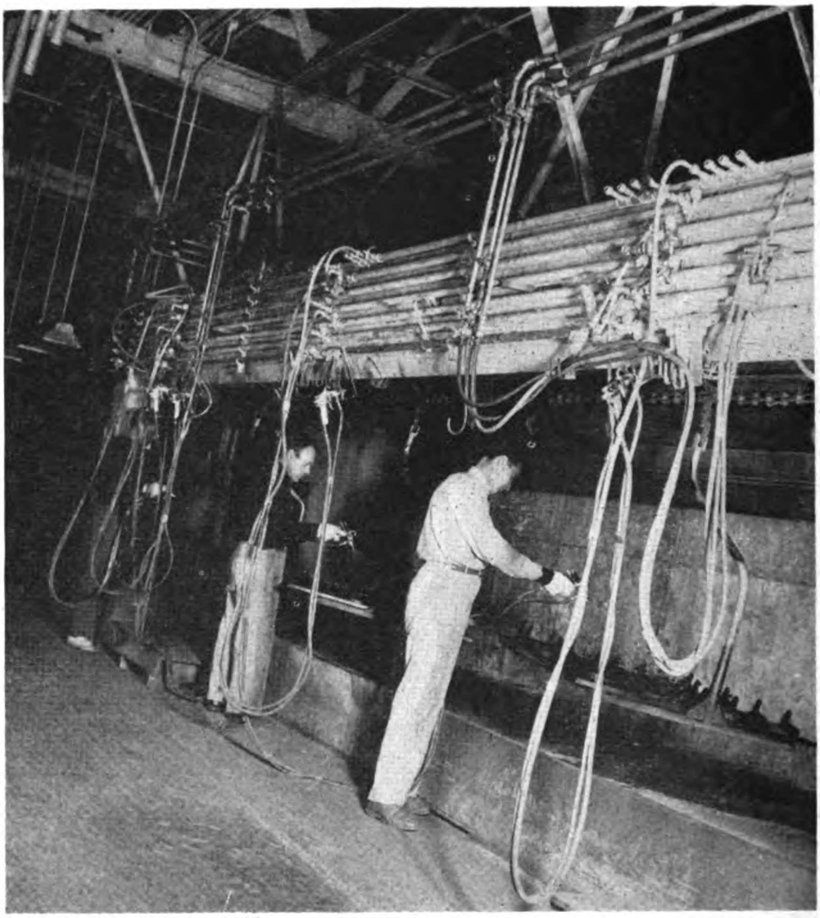


Fig. 108.—Spray paint booth with exhaust ventilation. Parts to be painted on moving chain.

and also increases the flexibility of the plant. Processes that are normally operated in conjunction with each other should be included in a single group, and if there are a few machines which are much in use they can be tied into two separate duct systems with blast gates arranged to throw the load to either one as dictated by the plant operations. Multiple exhaust systems may be served by individual dust collectors or one large collector may take care of several systems. In the latter case back pressure valves must be provided in each duct system to prevent back flow of air when one or more systems of the group are shut down.

Ducts should be located so that they are readily accessible, do not interfere with free access to the equipment served or to the operation of cranes, conveyors, elevators, trucks, etc., and where they will not be subject to injury by cranes, trucks, and other moving equipment. Locations conducive to the condensation of moisture inside the ducts are to be avoided. Underground duct locations are also to be avoided Ducts should be placed at least six inches above floors, where possible, and overhead ducts should be located close to the ceiling above.

Where large chips or heavy pieces of solid material may be carried along with the air stream into the duct system, it is advisable to install chip traps in the branch ducts, or such traps may be combined with the suction hoods. Chip traps, which depend on expansion only for their effectiveness, should have a cross-sectional area of at least eight times the area of the ducts served. The automatic dumping type of trap is not recommended due to danger of excessive air leakage.

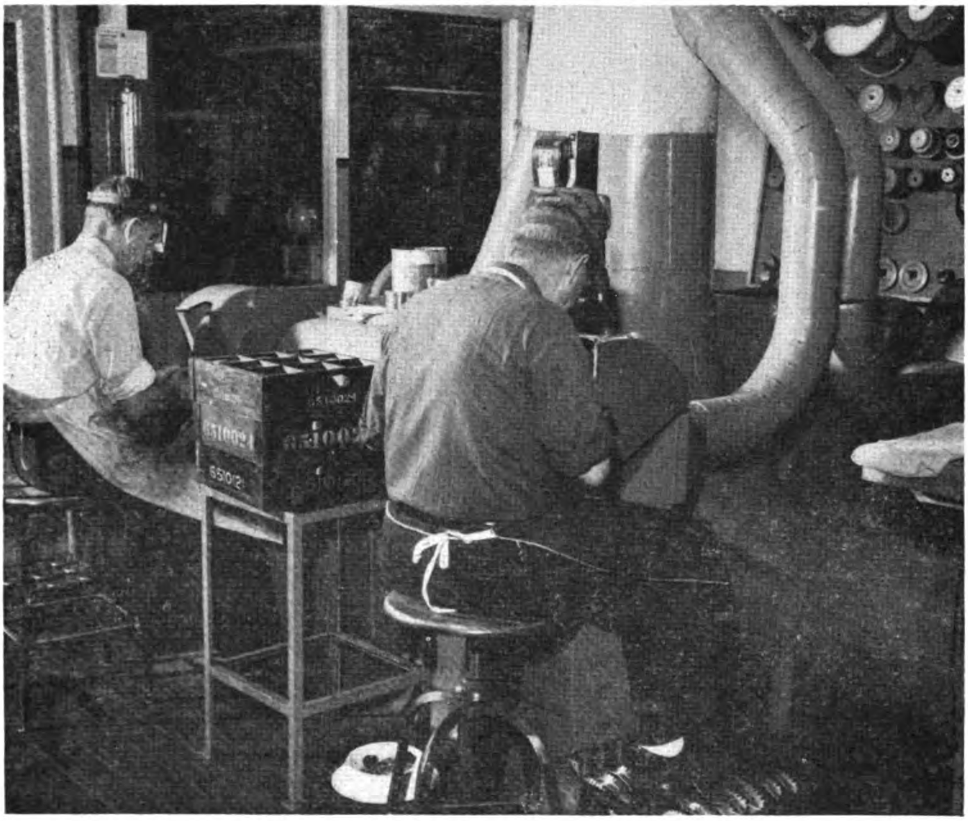


Fig. 109.—Buffers and polishers protected by local exhaust ventilation.

Ducts supplying return or replacement air should be located so that they do not cause excessive air motion near hooded processes. Return air ducts should also be arranged so that the workmen are not subjected to drafts of cold air.

Buffing and grinding wheel duct systems should be segregated, for sparks from the grinding wheels may set fire to the lint and grease from the buffing operation if both are carried through the same duct system (Figs. 109 to 112).

The duct layout should be arranged so as to avoid passing through fire walls or important floors. This can sometimes be accomplished by placing the main duct on the outside walls of the building. Frequently vertical risers can be located in stair wells. Insurance regulations require that where ducts do pass through fire walls they be provided with automatic fire dampers on both sides of the wall.

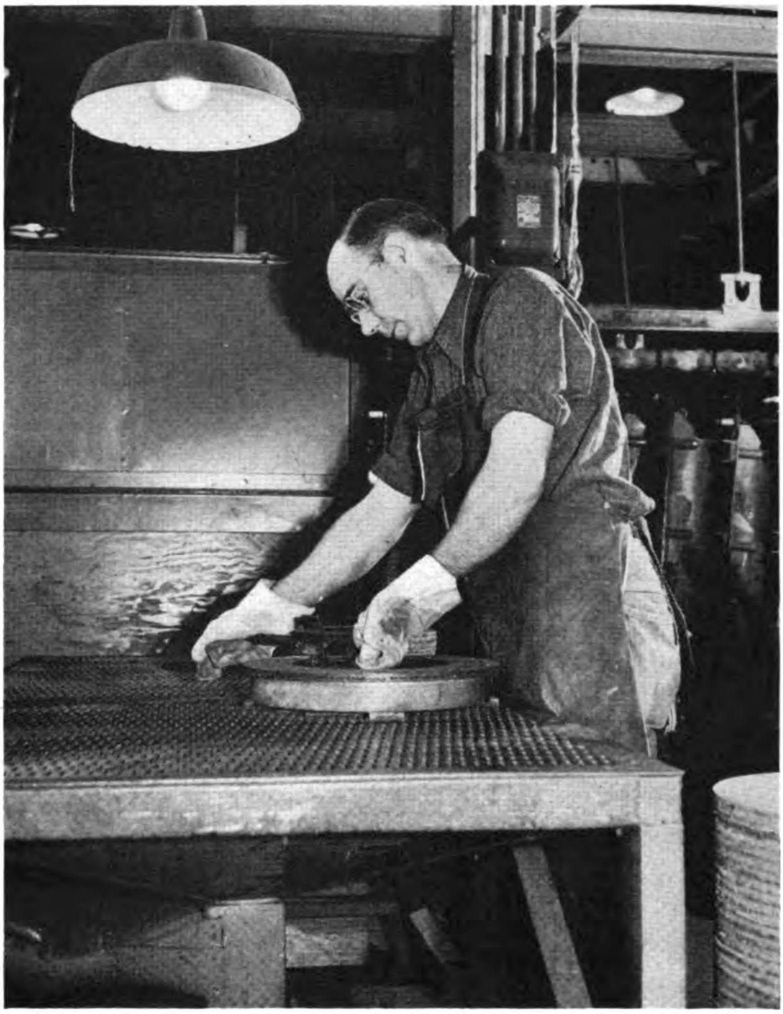


Fig. 110.—Dust particles drawn through perforated table—downdraft ventilation.

Duct systems for the removal of explosive or readily ignitable materials should be kept independent of other duct systems. The ducts should be as straight as possible and should lead directly to the outside of the building, never through other rooms. Automatic sprinkler heads should be provided near the inlet and outlet of the duct system. If installed inside the ducts they should be placed in an offset and not in the direct path of the air flow. Explosion relief vents are usually required on duct systems handling explosive or deadly flammable materials.

**Duct Sizes.**—Common practice in determining duct sizes is to make the branches the same size as the throat openings at the hoods, with the main ducts so sized that their areas at any point are between 20 and 25 per cent greater than the sum of the areas of the hood connections served between the point in question and the dead ends of the mains.

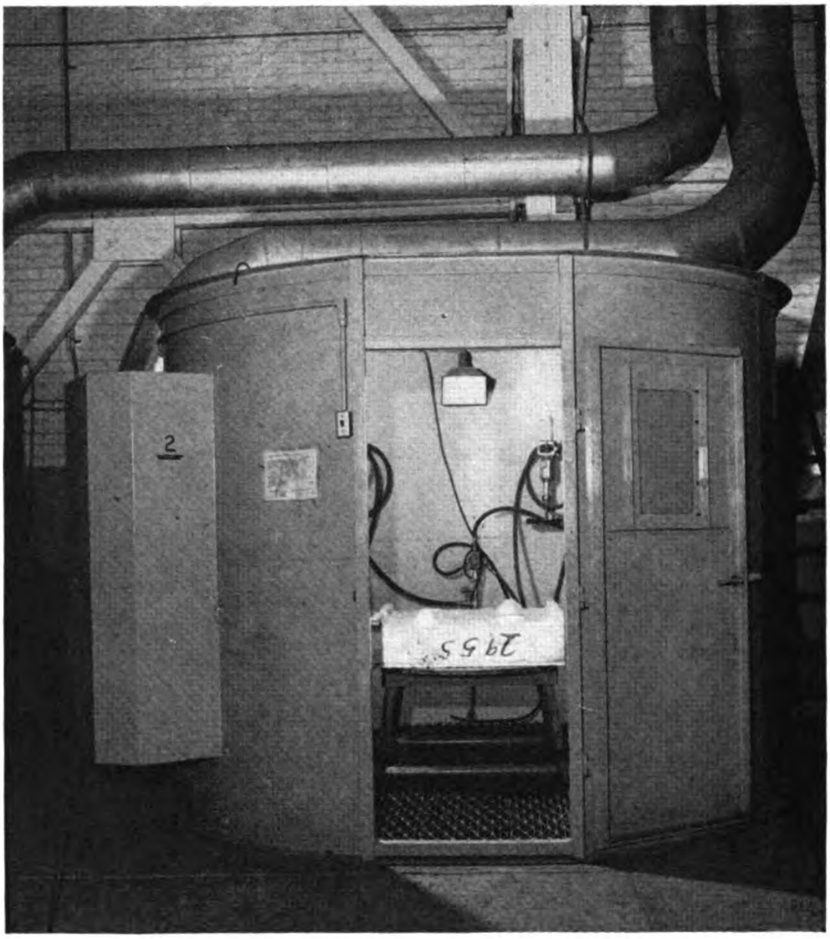


Fig. 111.—Grinding booth with downdraft ventilation. Note grill on floor.

The sum of the areas of the hood connections is known as "load area," and, on the basis of the above rule, branches are made equal in area to the load area which they serve. Submains and mains are sized so that at all points their areas are between 20 and 25 per cent greater than the load area served.

Since floor sweep ducts are used occasionally and for a few minutes at a time, they are not included in computing the load area or the size of the main ducts.

The method described of sizing the branches and mains in accordance with the load area is a development from certain state codes which specify the size hood connections and the minimum static suction at the hood connections for a given condition. These codes also limit the minimum duct sizes so that their area shall be not less than the connected load area, and for certain conditions they require areas 20 per cent greater than the connected load area (New York State code requires 20 per cent excess area in main ducts on systems used for removal of dust created by grinding, polishing, and buffing wheels). Usually the minimum static suction required at the hood connections is two inches of water. On the usual exhaust system, where the average restriction coefficient at the hood connections is taken as 0.7 and the ducts are sized on the basis of branches equal in area to the hood connections,

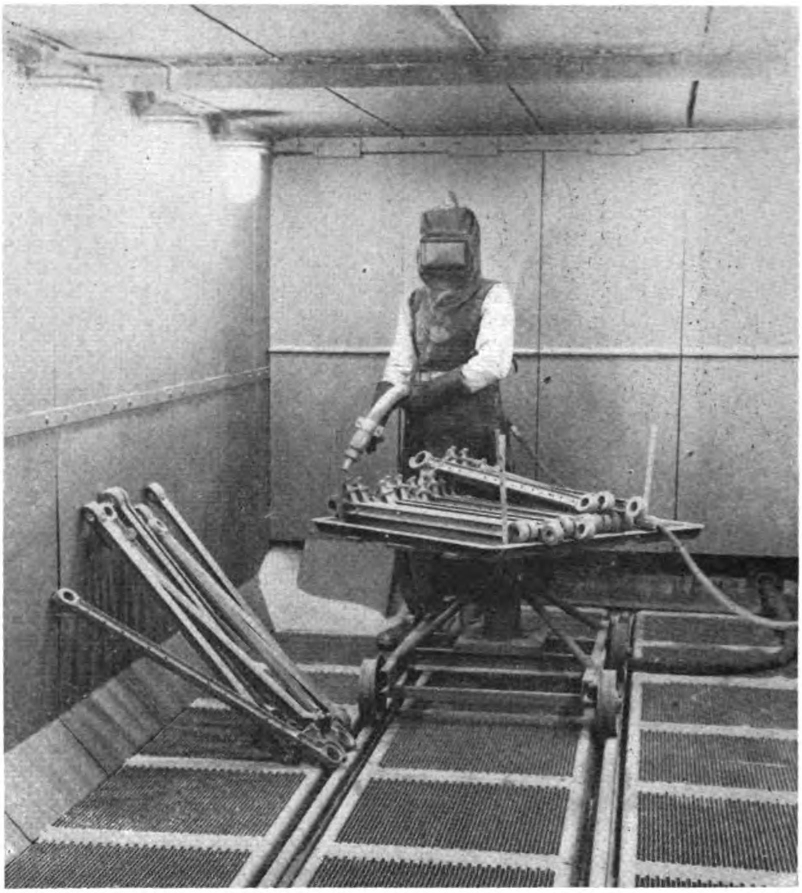


Fig. 112.—Sandblasting room with downdraft ventilation. Worker is likewise equipped with air supply sandblast helmet.

with mains 25 per cent greater area than the load area, the air velocity in the branches is 3960 feet per minute and in the mains, 3170 feet per minute. These velocities are sufficiently high to transport properly the dust and small particles collected by the exhaust hoods in metal grinding, buffing, and similar applications.

Improved operating efficiency and lower power consumption can be obtained by sizing the branch and main ducts so that they comply with the following basic requirements:

1. Area at each duct section should be such that the air velocity is not less than the minimum velocity required to carry the material exhausted from the hoods.
2. Maximum air velocities in branch and main ducts should exceed the minimum transport velocities only by the amount required to obtain even pipe sizes, with the exception that they should be increased in certain branches to obtain balanced suction at the hoods, as stated in paragraph 3.
3. In order to obtain balanced suction at the hoods, the pressure drop produced by the air flowing in any branch line should equal that obtained in that part of the main duct and branches which lies upstream to the branch under consideration.

**Transport Velocities.**—General practice is to design ducts for conveying dust from grinding and buffing wheels, tumbling barrels, sand-blast rooms, stone plants, and similar applications on a basis of a velocity of about 3,000 feet per minute. This velocity will also take care of dry sawdust, light wood chips, shavings, sander dust, etc. Grain dust, lint, cotton dust, and similar light materials are conveyed with 2,000 foot velocity. At the other end of the scale we have velocities of 4,000 to 4,500 feet per minute for wet sawdust and heavy wood chips, and of 5,000 feet per minute for lead dust.

Exact values of minimum transport velocities based on test data are meager, and there are usually unknown factors such as size and shape of particles, density, and lowered operating velocities under part load operating conditions. It can be stated as a general rule, however, that the required transport velocity increases with any increase in density of the material and it also increases with any increase in particle size.

**Conveying Capacity of Ducts.**—The quantity of solid material which can be conveyed in suspension in an air stream flowing in a duct is dependent upon the physical characteristics of the material, size of particles, density, and the volume of air flowing. Fine materials require more air than coarse materials. Heavy materials are moved with less air than low density materials. Experience has shown that with air velocities from 25 per cent to 50 per cent above the minimum transport velocities it requires 50 to 100 cubic feet of air to convey one pound of material.

Dust concentrations in exhaust ducts are usually much below the maximum and ordinarily it is not necessary to check the dust size against the quantity of material to be conveyed. However, in unusual cases, where the concentrations are high for any reason, as is the case where materials from several collectors are discharged into a separate exhaust system for transport to final destination, it is necessary to provide a duct of sufficient size to obtain an air flow of from 50 to 100 cubic feet of air per pound of material conveyed.

**Construction.**—Duct work for exhaust systems is usually fabricated from galvanized sheet iron, the gauges of which vary with the diameter of the duct and the suction of the fan. For low suction systems (up to 10 inches of water at the fan intake) the following gauges are recommended:

Small size ducts, 22 gauge
Ducts 16 to 24 inches in diameter, 20 gauge
Ducts 25 to 34 inches in diameter, 18 gauge
Ducts above 34 inches in diameter, 16 gauge

Exhaust ducts which handle large quantities of highly abrasive materials require heavier gauges. High suction systems also require heavier gauges or the ducts may be reinforced with band or angle iron.

Heavy-welded black iron ducts with flanged and gasketed joints are recommended for extremely abrasive conditions such as granite dust, etc., or where the ducts are subject to external injury or have to be taken down occasionally.

For corrosive conditions ducts may be of cast iron, lead, copper, stainless steel, monel metal, aluminum, tile, or other special material. Ducts may be lined with lead or rubber or painted with acid-resisting paints. Tile ducts are to be avoided due to danger of breakage and leakage.

Sheet metal elbows should be two gauges heavier than the ducts to which they are attached. The main trunks and branch ducts should be as short and as straight as possible. They should be strongly supported and protected against possibility of damage by moving equipment. The dead ends should be capped to permit inspection and, unless space will not permit, elbows should be long turn, with a center line radius equal to one and one-half times the diameter. Where a movable section of duct is required, flexible metal hose and ball or slip joints may be used.

## References

1. Design of Exhaust Hoods, Air Hygiene Foundation of America, Inc., Bull. No. 2, Part 4, April, 1938.
2. Hemeon, W. C. L.: Ventilation Requirements for Industrial Solvents, Heating and Ventilation, December, 1945, p. 97.
3. Design for Duct Work for Exhaust Systems, Air Hygiene Foundation of America, Inc., Bull. No. 2, Part 5, May, 1938.

# CHAPTER XL

# INDUSTRIAL HYGIENE—GENERAL—Cont'd

## Appraisal of Hazards

An essential function of industrial hygiene is to determine the presence and concentration of toxic materials in the workman's environment. The first step in this procedure is the collection of a sample of gas, air, or dust, and the second is the chemical analysis of the sample or, in the case of dusts, the counting of the dust particles.

In obtaining samples, several areas should be examined, such as:

1. The point of entrance of the contaminant into the atmosphere, *i.e.*, the source of the contaminant
2. The breathing zone of the worker
3. The immediate general vicinity of the worker
4. The general workroom atmosphere.

The samples are collected in vacuum tubes or vacuum bottles, gas or liquid displacement tubes, metal type containers, traps, bubblers, and absorbers according to the type of material to be sampled. Specialists in this field frequently design their own methods or improve upon those of others, so that there are numerous methods to be found described in the literature. Here again the reader is referred to the list of sources mentioned early in this section. Also the text by Jacobs[1] is highly recommended (Figs. 113 to 117).

Table XXII suggests the sampling method and the analytical method for determining the concentration of air contaminants. Goldman, in formulating this outline, emphasizes the fact that these methods are subject to improvement and that several of them are not entirely satisfactory.

**Dust Collection and Analysis.**—There are many methods of collecting samples of dust from indoor and outdoor atmospheres. From the standpoint of testing the industrial environment, the methods have narrowed down to electrostatic flocculation, filtration, and impingement. In the electrostatic flocculation method, the dust particles are caused to flocculate and thus precipitate by a high electric potential. A description of this method as well as descriptions of the Konimeter and Greenburg-Smith impinger methods can be found in the *United States Public Health Service Bulletin* Number 217 (1938). The Greenburg-Smith impinger samples air at a high velocity. By this method, air is impinged against a glass plate which is immersed in an absorption medium. The dust particles, momentarily arrested, are wetted and thus trapped. This impinger can also be used for sampling gases or liquids soluble in water or selected absorption media.

### Table XXII. Outline of Methods of Sampling and Analysis of Air Contaminants

(Prepared by Senior Chemist F. H. Goldman, Division of Industrial Hygiene, National Institute of Health, Bethesda, Md., March, 1943.)

| CONSTITUENT | SAMPLING METHOD | ANALYTICAL METHOD |
|---|---|---|
| Acetone | Bubbler with alkali: 5% sodium hydroxide | Iodometric |
| Acid fumes | Bubbler with<br>(a) water<br>(b) alkali (N/200 NaOH) | (a) Titration or determination of anion or pH determination<br>(b) Titration or determination of anion |
| Alcohol | Bubbler with 1.5% $K_2Cr_2O_7$ in 1:1 $H_2SO_4$ | Chromate reduction |
| Alkali mists | Bubbler with<br>(a) water<br>(b) acid (N/200 HCl) | (a) pH determination<br>(b) Titration |
| Ammonia | Bubbler with acid (N/200 HCl) | Nessler's reagent (Distill from alkali if necessary) |
| Ammonium chloride | Midget impinger | Nessler's reagent (Distill from alkali if necessary) |
| Ammonium nitrate | Midget impinger | Phenoldisulfonic acid |
| Ammonium picrate | Midget impinger | (a) Colorimetric<br>(b) Distill from alkali: Nessler's reagent |
| Aniline | Fritted bubbler with 1:100 $H_2SO_4$ | Colorimetric with $CaOCl_2$—phenol |
| Antimony dusts | Midget impinger | (a) Colorimetric Rhodamine B<br>(b) Spectrographic |
| Arsenic dust | Impinger | Gutzeit |
| Arsenic fumes | Electrostatic precipitator | Gutzeit |
| Arsine | Test paper with mercury bromide | Visual |
| Benzol | (a) Indicator<br>(b) Mercury seal flask<br>(c) Sampling bottle with 2 ml. sulfuric-fuming nitricacid<br>(d) Special bubbler with nitrating acid ($HNO_3 + H_2SO_4$) | (a) Visual<br>(b) Absorption spectrum<br>(c) Colorimetric or titration with titanous chloride<br>(d) Colorimetric or titration with titanous chloride |
| Cadmium | (a) Impinger<br>(b) Precipitator | Cadmium sulfide or dithizone |
| Carbon bisulfide | (a) Bubbler with 5% KOH<br>(b) Bubbler with diethylamine copper acetate reagent at 60 ml. per minute | (a) Ethyl xanthate<br>(b) Colorimetric; may be compared immediately without any additional chemical operations |
| Carbon monoxide | (a) Indicator<br>(b) Sampling bottle | (a) Visual<br>(b) Iodine pentoxide |

Unless otherwise stated sampling rates are:

| | |
|---|---|
| (a) Impinger | 1 cubic foot per minute |
| (b) Midget impinger | 0.1 cubic foot per minute |
| (c) Bubbler | 0.5 to 1 liter per minute |

In cases where homogenous solutions are obtained due to solubility or reaction of the constituent with the sampling media, rinsings should *not* be combined with the sampling media but the *volume* of sample liquid should be recorded. This avoids unnecessary dilution of the sample.

In cases where the midget impinger is used with media other than water, it is preferable and sometimes absolutely necessary to use all-glass apparatus. If rubber must be used, then samples should be transferred to all-glass containers immediately after sampling.

The collecting medium is water unless otherwise stated.

Where fritted glass bubblers are used, the porosity is *coarse.*

TABLE XXII—CONT'D

| CONSTITUENT | SAMPLING METHOD | ANALYTICAL METHOD |
|---|---|---|
| Chlorine | Bubbler with o-tolidine solution | Colorimetric (Immediately in the field) |
| Chlorine dioxide | Bubbler with 5% KI solution | Iodometric |
| Chlorinated hydrocarbons | (a) Two bubblers in series with amyl acetate<br>(b) Halogenated combustion apparatus<br>(c) Silica gel | (a) Chemically after burning<br>(b) Volumetric Volhard or gravimetric AgCl<br>(c) Chemically from silica gel |
| Chromic acid mists | Impinger with alkali (5% NaOH) | (a) Iodometric<br>(b) Colorimetric |
| Cyanide | Bubbler with alkali (5% NaOH) | (a) Volumetric } Best imme-<br>(b) Colorimetric } diately |
| Dimethyl aniline | Fritted bubbler with 1:100 $H_2SO_4$ | Colorimetric as the nitroso derivative |
| Diphenylamine | Fritted bubbler with 1:100 $H_2SO_4$ | Colorimetric with Fe |
| DNT (dinitrotoluene) | (a) Fritted bubbler or impinger with diethylaminoethanol<br>(b) Fritted bubbler or midget impinger with alcohol | Colorimetric |
| Esters | (a) Vacuum bottle<br>(b) Charcoal tube<br>(c) Benzol indicator | (a) Iodine pentoxide<br>(b) Gravimetric<br>(c) Visual (special calibration) |
| Ether | (a) Benzol indicator<br>(b) Bubbler with alcohol | (a) Visual (special calibration)<br>(b) Volumetric after hydrolysis |
| Fluorides<br>(a) Dusts<br>(b) Fumes<br>(c) Vapors | <br>(a) Midget impinger<br>(b) Electrostatic precipitator<br>(c) Fritted bubbler | Titrate with thorium nitrate or colorimetric |
| Formaldehyde | (a) Impinger with 1% sodium bisulfite<br>(b) Impinger with 1.25% KOH | (a) Volumetric estimation of sulfoxalate formed<br>(b) Polarigraph or colorimetrically by Schryver method |
| Hydrogen sulfide | Bubbler with cadmium chloride | Iodometric |
| Lead | (a) Impinger with 1:100 $HNO_3$<br>(b) Electrostatic precipitator | Colorimetric—dithizone<br>Chromate (gravimetric) |
| Lewisite | Fritted bubbler with 5% NaOH | Chemical determination of chloride |
| Magnesium | Midget impinger | Spectrographic |
| Manganese | (a) Impinger<br>(b) Precipitator | Colorimetric |
| Mercury | (a) Detector<br>(b) Midget impinger | (a) Visual<br>(b) Colorimetric by dithizone |
| Metal fumes | Electrostatic precipitator | Chemical or spectrographic |
| Methanol | Bubbler with 5% alkaline permanganate | Colorimetric |
| Methyl butyl ketone (butanone) | Bubbler with 5% NaOH | Iodometric |
| Methyl cellusolve | Bubbler with 1.5% $K_2Cr_2O_7$ in 1:1 $H_2SO_4$ | Colorimetric or iodometric |
| Mono-oil (Mononitrotoluene) | Gas sampling tube and 2 ml. nitrating acid | Colorimetric or titration with $TiCl_3$ |

*(Cont'd on next page)*

TABLE XXII—CONT'D

| CONSTITUENT | SAMPLING METHOD | ANALYTICAL METHOD |
|---|---|---|
| Mustard gas (Dichlorodiethylsulfide) | Fritted bubbler with 5% alkali | Determine chlorides after hydrolysis |
| Naphtha | (a) Benzol indicator<br>(b) Charcoal | (a) Visual (special calibration)<br>(b) Gravimetric |
| Nitrobenzene | Sampling bottle with 2 ml. nitrating acid | Colorimetric or titration with $TiCl_3$ |
| Nitroglycerin | Fritted bubbler with triethylene glycol | Colorimetric with xylenol |
| Nitrogen oxides | Sampling bottle with 10 ml. N/10 $H_2SO_4$ with 3 drops 3% $H_2O_2$ | Colorimetric with phenoldisulfonic acid |
| Pentaerythritol | Midget impinger with 1.5% $K_2Cr_2O_7$ in 1:1 $H_2SO_4$ | Colorimetric |
| PETN (Pentaerythritol tetranitrate) | Fritted bubbler with triethylene glycol | Colorimetric with xylenol |
| Phenol | Bubbler with 5% NaOH | Colorimetric |
| Phosgene (Carbonyl chloride) | Bubbler with 5% NaOH | Chemically for chlorides |
| Phosphine | (a) Bubbler<br>(b) Test paper with $AgNO_3$ | (a) Chemically<br>(b) Visual |
| Potassium chlorate | Impinger | Volumetrically with $FeSO_4$ and $KMnO_4$ |
| Propylene glycol | Bubbler | Iodometric using periodate reagent |
| Solvent vapors | Benzol indicator | Visual (Special calibration) |
| Sodium oxalate | Fritted bubbler | Chemically: titration with $KMnO_4$ |
| Strontium salts | Impinger | Spectrographic |
| Sulfur chloride | Bubbler with 5% alkali | Chemically: Volumetric |
| Sulfur dioxide | Bubbler with 5% alkali | Chemically |
| Tetryl | (a) Midget impinger with 1:100 $H_2SO_4$<br>(b) Midget impinger with diethylaminoethanol | (a) Colorimetric with alkali<br>(b) Colorimetric |
| Toluol | (a) Benzol indicator<br>(b) Mercury seal flask<br>(c) Sampling bottle with 2 ml. nitrating acid ($HNO_3 + H_2SO_4$) | (a) Visual (Special calibration)<br>(b) Absorption spectra<br>(c) Colorimetric or $TiCl_3$ titration |
| Triethylene glycol | Bubbler with $K_2Cr_2O_7$ in 1:1 $H_2SO_4$ | Colorimetric or volumetric |
| TNT (Trinitrotoluene) | (a) Midget impinger with alcohol<br>(b) Midget impinger with diethylaminoethanol | Colorimetric |
| Zinc | Precipitator or impinger | Chemically |

Dust particles which are collected by various means are counted, measured and estimated gravimetrically. Greenburg and Bloomfield recommend the following procedure for counting dust particles:

As soon as practicable and preferably before twenty-four hours have elapsed because dust particles are slightly soluble in water, the stopper of the flask in which the sampling was done is removed and carefully washed. The washings are added to the contents of the flask. The entire sample is filtered into a volumetric flask through a screen of approximately 325-mesh fineness. This will eliminate all particles over 40 microns, for only those smaller than that will go through the filter. If the dust suspension in the volumetric flask is too dense, it should be transferred to a larger volumetric flask and diluted further, or an aliquot should be removed and diluted to a known volume. The dilution should be continued until the number of particles counted in each microscopic field is about 50 to 75. Agitate the contents of the volumetric flask so that a uniform suspension is obtained. Remove two 1 cc. aliquots with a 1 cc. pipette and just fill two Sedgwick-Rafter counting cells.

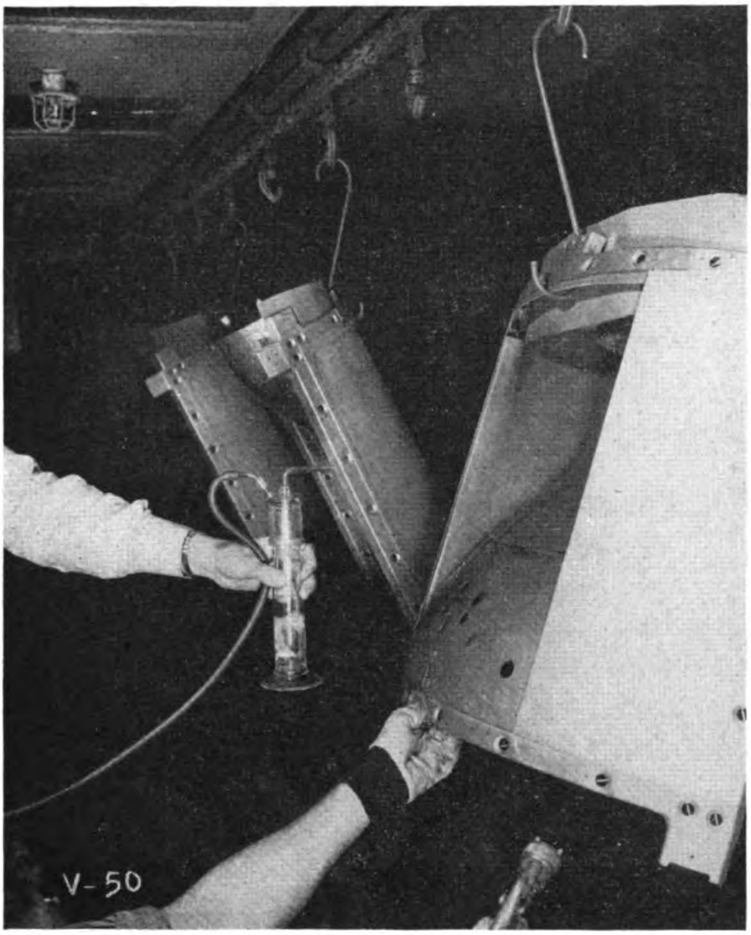


Fig. 113.—Sampling air in the breathing zone of the worker.

A Whipple ocular micrometer disk, or a modification, should be used as the eyepiece. This disk has a large square engraved on it, covering a large part of the field, and this square is divided into 100 medium-sized squares. One of these is in turn further subdivided into 25 small squares. Using an ordinary microscope provided with a suitable eyepiece and objective and fitted with an Abbe condenser, the proper tube length of the microscope is determined by calibration with a stage micrometer, so that the side of the large square of the eyepiece covers 1,000 microns (1 mm.). The large square of the eyepiece ruling, therefore, incloses the dust in an area of 1 sq. mm., and since the cell is 1 mm. deep, all the dust suspended in 1 c.mm. of the water is under the ruled field. This examination is accomplished by raising and lowering the lens system so as to focus throughout the entire depth of the cell. As a source of illumination, an ordinary small electric microscopic lamp may be used.

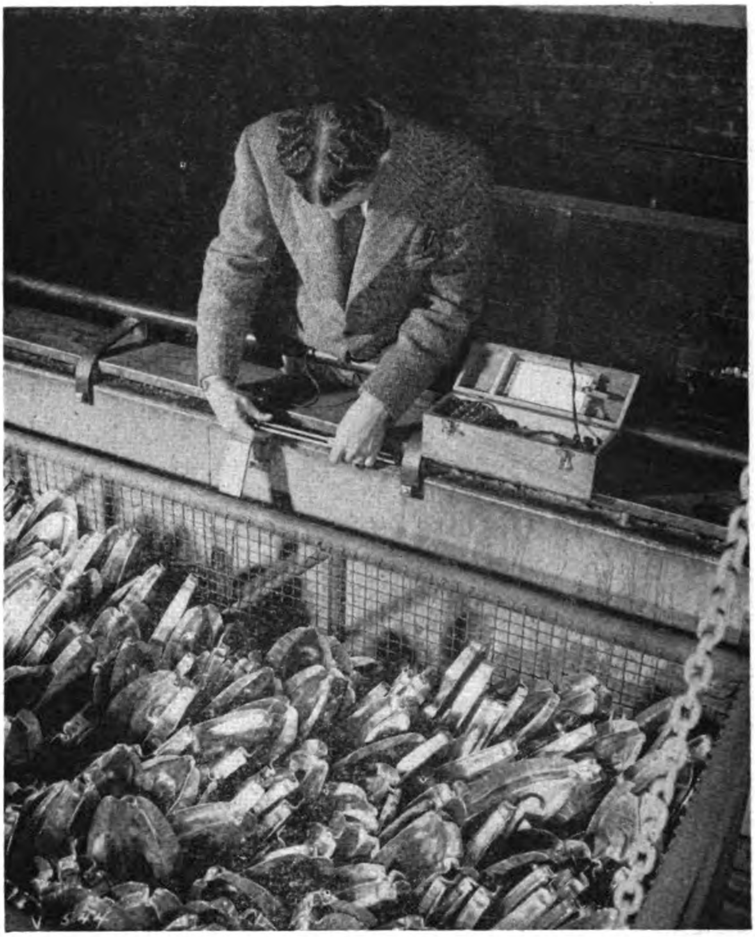


Fig. 114.—Sampling air at the level of the lateral exhaust ventilation.

The dust is permitted to settle for twenty minutes before counting is done. In general, only particles less than 10 microns in diameter are counted. The inclusion of particles larger than 10 microns makes very little difference in the total count. The average diameter of a particle for the purpose of this exclusion is judged by inspection. In practice it is necessary to count the dust in only one-quarter of each ruled field, the entire field having been examined for uniformity. Such counts on five fields, so dispersed as to be representative, are made on each of the two Sedgwick-Rafter cells. These ten counts are averaged, but this average is not to be taken as the final count until a corresponding control count has been subtracted. In all cases a sampling flask which is handled in the plant, but through which no air has been aspirated, is used as the control for the particular series of samples taken in that plant on that particular day, and counts are made on this control fluid in the same manner as on the fluid through which the air sample has been impinged. The control sample takes into consideration any dust which may be present in the eyepiece micrometer, in the lenses of the microscope, in the Sedgwick-Rafter counting cell, and in the sampling liquid itself.

Fig. 115.—A modern hygiene laboratory in an industrial plant.

From the average gross count obtained on the impinger sample, the average control count is to be subtracted to give the average net count per one-quarter microscopic field.

The average net count per one-quarter microscopic field is multiplied by four, to yield the average count in the total field. Since the Sedgwick-Rafter cell is 1 mm. deep, this figure represents the number of particles in a cubic millimeter of the diluted sample. This value is multiplied by 1,000 to give the count per cubic centimeter of sample and again by the total number of cubic centimeters of liquid to which the original sample was diluted. This product is divided by the number of cubic feet of air sampled.

Fig. 116.—Preparation of material for counting dust particles.

To summarize, the number of particles per cubic foot of air equals the average net count per one-quarter field multiplied by a factor, where the factor equals

$$\frac{4 \times 1000 \times \text{total volume of diluted sample in cc.}}{\text{Volume of air sampled in cubic feet}}$$

The average dust exposure of a worker is ascertained by multiplying the average dust concentration obtained in the count in each activity by the time spent in the amosphere.

In determining the size of the dust particles, a filar micrometer ocular and oil immersion objective with a magnification of 1,000

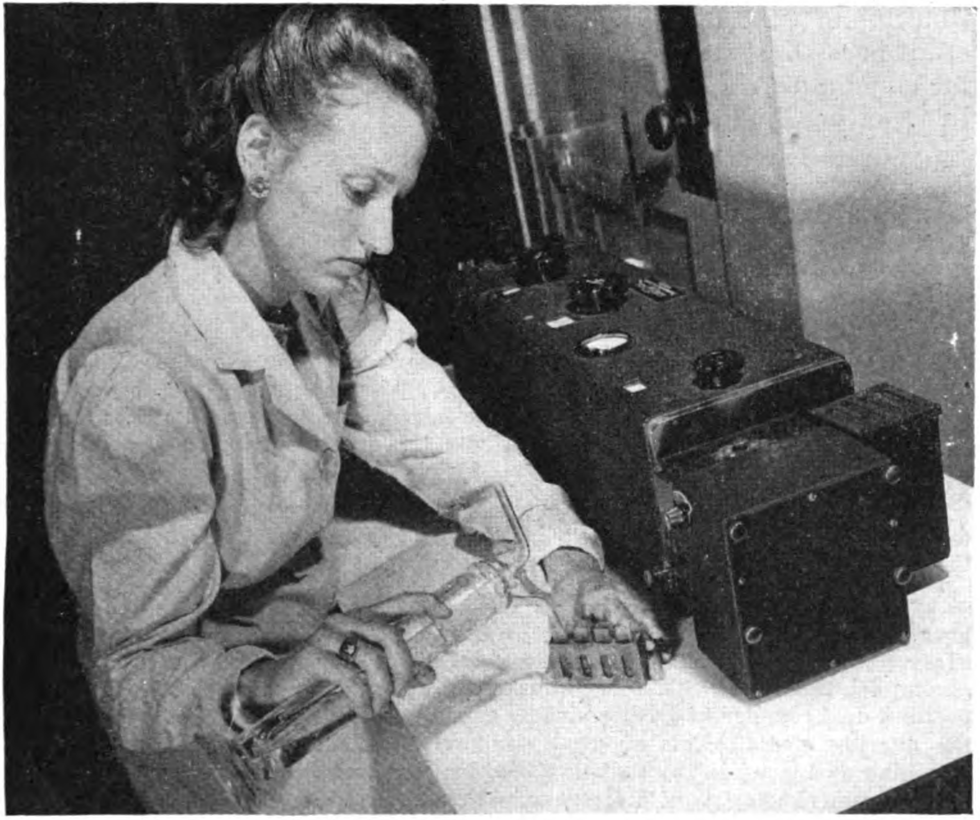


Fig. 117.—Electrocolorimetric determination of a collected sample.

diameters are used. With this magnification, particles as small as 0.5 micron can be measured, while smaller particles can be distinguished and recorded. The filar micrometer eyepiece is calibrated so that one division on the micrometer represents a known length on the stage of the microscope. After the microscope is focused upon a representative section of the sample, the horizontal diameter of 200 particles is measured with the filar micrometer, moving the cross-hair across the field from left to right and measuring the first 200 particles encountered. This is done in several representative fields. The sizes of the particles are recorded in terms of divisions of the filar micrometer and may later be converted into microns by means of a calibration factor. Only one dimension of the particles need be measured, since the particles lie in the field at random and it is impossible to rotate the stage of the microscope at times. Because, however, a large number of particles can be measured, if desired, the results are the same as if the average of the length and breadth were determined for each particle.

In the diagnosis of silicosis it is imperative in most instances to determine the particle size of the dust which constituted the exposure. Holden and associates[2] proposed a method which would secure samples of air-borne dust which could be fractionated by size so as to determine the amount of free silica in the sizes which are known to be dangerous. In reporting this new method, Holden and co-workers stated:

> "For several years it has been our practice to employ the electrostatic precipitator for the collection of samples of dust for composition analysis, collecting them from the breathing zone of workmen. The analysis of these samples, however, consistently showed the presence of dust particles as large as 50 microns or even larger. Analysis for free silica is always reported on a per cent by weight basis. Therefore, the presence of one 50 micron particle would have *as much effect on the analysis* as would 1,000 five micron particles.
>
> "It was therefore logical to modify our techniques to include separation of the air-borne dust samples into two size fractions. We chose to separate the dust into portions at the five micron level. The five micron and less dust is designated as respirable dust, in the sense that it is only this fraction that reaches the lung depths, and its analysis provides the data for appraisal of the silica dust exposure.
>
> "In earlier work Hatch and Moke reported that 73 per cent of the weight of dust in foundry samples was contributed by particles over 10 microns, whereas these amounted to only one per cent of the total number.
>
> "The satisfactory application of size separation required quantities of air-borne dust amounting to several hundred milligrams. In some instances it was necessary to operate the electrostatic precipitator during several work days to accomplish this result. This was impractical. Accordingly, we developed a composition dust sampler of greater airflow capacity, which employs salicylic acid as the filtering media. This material was first suggested by British workers for obtaining dust samples from air of mines for chemical and petrographic analysis. Our instrument consists of a rectangular box whose sides consist of interchangeable double screens. The box is 3 inches wide and 10 inches square. There is a one-inch opening at one end from which air can be drawn through the filter. Each side of the filter is made from two 100-mesh copper screens which serve as supports for a ⅛-inch layer of salicylic acid crystals. A number of sets of these filter screens have been provided for the instrument so that they can be filled in the laboratory, attached together, and transported for field work. It is necessary to make certain that there are no large openings in the salicylic acid filter mat. The presence of large openings will reduce the efficiency of filtration, requiring longer periods of time for the collection of the sample.
>
> "Air from the breathing zone of workmen is drawn through the filter, employing a small blower. We have modified our electrostatic precipitator so that it can be used as a source of suction, drawing approximately 20 cubic feet of air per minute. The electrostatic precipitator with the I-H-F filter will therefore collect as much dust in one hour as the precipitator alone will collect in one day. Two other blowers which we commonly use with the filter draw 50 and 56 cubic feet of air, respectively.

"Salicylic acid resembles macerated blotting paper. However, it will dissolve completely in alcohol leaving the dust suspended. The dust is filtered from the alcohol on hardened filter paper (No. 50 Whatman) in a Buechner funnel employing suction. It is necessary to wash the dust until all the salicylic acid is removed. The dust is then washed from the paper with alcohol and separated into size fractions.

"The size separation is accomplished by sedimentation of the coarse dust. This is done by a repeated settling procedure with the fines being removed by suction at each time interval. We settle the coarse dust from a 3.3 centimeter layer of alcohol for 40 minutes to separate the five micron level.

"A multiple unit for this purpose will size fraction four samples at one time."

## References

1. Jacobs, M. D.: The Analytical Chemistry of Industrial Poisons, Hazards and Solvents, New York, 1941, Interscience Publishers, Inc.
2. Holden, F. R., Hemeon, W. C. L., and Hyatt, E. C.: Accurate Measurement of Silicosis Hazards; Newer Methods, Industrial Hygiene Foundation Bulletin, Transaction Series, No. 6, 1946.

# CHAPTER XLI

# PRE-EMPLOYMENT EXAMINATIONS AND THE PLACEMENT PROGRAM

As now conducted by the average industry, the pre-employment examination is a useless and economically wasteful procedure which fails to benefit either the employer or the applicant seeking employment. Originally, the pre-employment examination was instituted to detect physical defects in applicants in the belief that if the physically defective were denied employment, the accident rate would be lessened and thereby reduce the cost of claims for workmen's compensation. This plan failed since proneness to accidents is not necessarily synonymous with physical defectiveness.

Having learned this lesson, however, large plants did not discard the pre-employment examination plan, but rather used it as a guide for the placement of applicants seeking employment. Also, many of the larger companies who were earnestly attempting to carry out a program of good preventive medicine wisely felt that the pre-employment examination was the initial step in order to accomplish such an aim.

Unfortunately, the smaller industries, lacking direct medical supervision, have not extended the use of the pre-employment examination to this intelligent and productive end. As a matter of fact, it is doubtful if the majority of small industries would ever have instituted the pre-employment examination if they had not been sold the idea by certain well-meaning agencies. Management "bought" the program much as it buys fluorescent lighting or drinking fountains. Once installed, management assumed that the program would work automatically, without requiring any further attention. If the usual practice of conducting the pre-employment examination is analyzed, two primary deficiencies in the prevailing procedure will be noted.

1. The forms used for the pre-employment examination

A survey of a large group of plants revealed that the examination blank in use was not designed by the management of that plant or by a physician connected with that plant; the blank did not even consider the specific occupations carried on within that plant. Invariably the forms in use had been copied from some other company and were standardized as the traditional check book. It was found that a few companies used a blank supplied by the examining physician and that the form was the same as the one which the physician used in examining applicants for employment for many other and often dissimilar industries.

A particularly bad practice noted in the survey was the frequent use of the short form, the "two dollar" examination blank. Such forms call for the most cursory type of examination, have inadequate space for physical findings, and no room for detailed history. For management's investment of two dollars in such an examination it certainly receives only two dollars worth of information—if that much.

2. The selection of the examining physician

Our survey revealed that in those industries which did not employ a full-time physician, pre-employment examinations were made by: (1) the "uptown" doctor who served this company in no other capacity; (2) the neighborhood physician who cared for the plant's medical and surgical emergencies in his office; or (3) the applicant's family physician. Any of these three methods renders the pre-employment examination a detached procedure, for, with rare exceptions, the physician chosen in this manner is not acquainted with the hazards residing in that particular industry, nor with the requirements of the occupation to which the applicant is to be assigned.

In order to make an industrial health program effectual, one of the first measures should be the correction of the existing inadequacy of the pre-employment examination. This can be done by advising management through channels related to industry as to the type of examination best suited to individual plant needs and by educating the medical profession as to the true objective of industrial examinations.

For ordinary types of employment the physical examination should be routine but complete. The author has been intimately acquainted with the excellent medical program at Lockheed Aircraft Corporation, devised and directed by Dr. Fenn E. Poole. Under his guidance, more than 300,000 employees were examined, of whom more than 15,000 physically substandard workers were successfully placed. In describing his objectives and methods, Poole stated[1]:

> "Generally, it is sufficient to determine that the worker has normal vision, hearing and use of the extremities and is free from communicable disease, serious disease of the heart or the lungs, hernia, deformity, or any other condition which would be aggravated by employment. Depending on the needs of the specific industrial plant, this basis examination may be expanded in many ways; for example, in a plant where high noise levels are encountered, closer attention must be given the ears, while in a factory where much heavy lifting is done, special attention must be paid to the body structure, back and hernias."

The applicant's history parallels in importance the physical examination. The examining physician must keep uppermost in his mind that he is engaged in a "selection operation." Even if the physical examination may reveal no evidence of allergic tendencies, the history should; or the examination may reveal no evidence of past disease of the lungs, kidneys, or liver, but the history should. Therefore, despite normal findings at physical examination, the applicant should not be assigned to an occupation which might aggravate or reactivate a disease of which the history alone indicated the previous existence.

The pre-employment examination should include a laboratory investigation consisting of urine analysis, blood count, and chest x-rays. From the standpoint of preventive and curative medicine, the Wassermann is also desirable, but it has little value in the industrial examination. Further tests should be carried out if indicated through the special requirements of the employment.

What has been written regarding pre-employment examination also applies in most respects to the periodic re-examination. The scope of such a check-up should not be too limited. There is a tendency in industry to recheck men engaged in hazardous occupations by repeating laboratory tests which will reveal undue absorption of toxic substances, or to test for possible dysfunctions of certain systems which might impair the worker's efficiency for his specific job. For instance, a crane operator had his vision and blood pressure checked every six months. One day he dropped a load of steel rails upon a fellow worker, because he suffered a diabetic coma. For some years the urine had never been examined. Blood lead determinations should be done every four to six months, and smears for basophilic stippling still more frequently, depending upon the type of exposure to lead. For those in dusty trades, the chest x-rays should be repeated at six-month intervals. Those in noisy trades need an initial audiogram and repeated audiograms at four- to six-month intervals. Other tests for those working with solvents must be repeated at short intervals, and the visual acuity of those for whom vision is a paramount need requires periodic checking.

There is also no reason why periodical examination should apply only to the plant personnel. It should be mandatory for all executives and office workers. A good industrial health program should recognize no line of demarcation between the plant and the front office.

**The Job Placement Program.**—It has been pointed out that from poorly conducted pre-employment examinations, the employer and the employee receive only negative values. The positive values to be obtained from a good pre-employment procedure are those which evaluate the applicant's capacities and utilize these by proper occupational assignment. This concept has but recently supplanted the previous system of rejecting every applicant who evidenced a physical defect of any type.

Following World War I, considerable thought was given to the project of rehabilitating the war wounded, but the same degree of endeavor was not expended to rehabilitate the industrially injured, despite the efforts of Kessler for such a program. Early in World War II it became evident that if America was to avoid defeat it would have to utilize the men and women who could not qualify for military service.

To this end the aged, the blind, the crippled, the epileptic, the cardiac, and all those who had heretofore been classified as unfit for work in industry were called upon to win the war on the home front. That these presumably unfit individuals did an efficient job needs no further testimony than the statistics of unbelievable production.

Due to extensive investigations by such men as Hanman,[2] Kuh,[3] Bridges,[4] Harvey and Luongo,[5] and many plant medical directors who must remain unnamed, a sane and practical method of job placement was evolved. The physical requirements of a myriad of jobs were analyzed and the environmental factors of each were listed. Following the study of the job requirements, a plan was suggested in order to match men and jobs. All of the various studies emphasize that the evaluation of the applicant's physical capacities is the obligation of the physician, while the job analysis placement procedures are technical features of the program.

In the preface to his original work, Hanman[2] clearly stated the objectives of the job placement program:

> "In developing personnel relations to the greatest satisfaction of the worker, the employer, and the community, *selective placement* is that process whereby the worker is chosen and assigned to that job (1) which will afford the greatest utilization of his skills and aptitudes, and (2) which at the same time will be compatible with his physical capacities and protect his health and safety. These two steps in the selective placement process may be referred to more readily as (1) matching the *skill characteristics* of workers and jobs, and (2) matching the *physical characteristics* of workers and jobs.
>
> "Existing techniques for relating *skill characteristics* are relatively adequate when compared with methods for matching *physical characteristics*. It is believed, therefore that the emphasis should be placed on the development of better techniques for relating the *physical capacities* of workers to the *physical demands* for jobs. Evidence of this emphasis is to be found in the many and varied efforts of industry and government to find a solution to the *physical* aspects of the selective placement problem. The primary motivation for this emphasis is based on the desire to make it possible to place all workers successfully and safely on jobs in accordance with their physical capacities, particularly disabled veterans."

The technique developed by Hanman, and the many charts which describe the use of his technique, cannot be reproduced here, but the reader can find these in the publication referred to.[2]

An excellent approach to the problem is found in the book by Bridges.[4] Neville Pilling summarizes this work, a portion of which reads as follows:

> "Few persons are physically perfect. Most people have some physical defect —some deviation from normal that affects their manner of living and working. It may be only a minor defect, such as flat feet or color-blindness; or it may be a major and more serious defect. It may be obvious and understandable, such as a crippled hand or a missing leg; or it may be hidden and not readily understood, such as epilepsy or heart trouble or diabetes. Or it may be a borderline or transi-

tory defect such as an allergy or migraine headache, which may require special consideration upon occasion because it causes fatigability or distracting physical discomfort and thus may lead to accidents.

"Though the average person may have one or more of these defects, he probably has never considered himself 'physically handicapped,' 'disabled,' 'impaired,' or 'crippled.' This is because there is no common agreement or accepted definition of these terms. To most people they mean an obvious and very serious defect. This connotation probably has been largely responsible for the widespread failure to realize that most handicapped persons are only partially disabled and have a far greater amount of *residual ability* than *disability*. To date no one has been able to devise a term that adequately expresses the thought 'much ability-little disability.' The best suggestions have been 'limited' or 'partially limited.'

"From the industrial standpoint, however, anyone is physically handicapped who requires selective job placement because of a physical or mental impairment. In industry any deviation from 'normal' may be considered a physical disability, if it:

"1. Requires a person to modify or change his occupation.

"2. Makes it more difficult to obtain employer acceptance for suitable employment.

"3. Requires special consideration to avoid situations or working conditions that may aggravate the disability or jeopardize the health or endanger the safety of the person or his fellow workers.

"In other words, a defect is considered a physical disability, industrially, if it creates certain work limitations—if there are industrial activities which the person cannot or should not undertake."

**Elements in Successful Placement.**—Successful, correct placement involves an understanding of several fundamental principles, most important of which are the following:

1. Nearly all disabled persons have more ability than disability.
2. Very few jobs require all of a man's ability.
3. Proper placement involves matching a worker to a job on the basis of his *ability* to meet the demands of the job. When this is done, the disability disappears as a job factor.
4. It is important to understand the nature of a disability and what it means in terms of work limitations and health and safety considerations. In other words, each disability may impose upon the individual certain limitations as to the activities in which he may engage and the working conditions and accident and health hazards to which he may be exposed.
5. It is possible to employ suitably the great majority of workers classifiable as handicapped, but there is a small percentage who are employable only under sheltered conditions or in special workshops. There are also some who are homebound and are not classifiable as employable except under special arrangements.

Whatever a person's disability may be, to match him successfully with a job in which his health and safety will be maintained and in which his efficiency will equal or excel that of an able-bodied person, these four requirements must be met:

1. *Physical ability:* The worker should have the ability to accomplish the task efficiently, *i.e.*, be able to meet the physical demands of the job. For example, a man on crutches could not be placed in a job requiring heavy lifting, carrying heavy weights, or extensive walking, especially on slippery surfaces.
2. *Personal safety:* The worker should not be a hazard to himself. For instance, a person subject to dizzy spells should not work on a ladder or scaffold or around moving machinery, where he may be seriously injured or killed if he becomes dizzy on the job.
3. *Safety of others:* A person should not work at a job where his disability is likely to cause an accident that might injure others. An epileptic person, for example, should not drive a bus or operate an overhead crane, because he may have an attack while working, lose control, and cause injury to others.
4. *Personal health:* The job should not aggravate the disability of the worker. For instance, a person with heart trouble should not be placed in a job that requires considerable stair climbing, running, heavy lifting, or other strenuous tasks. A person with a skin disease should not be exposed to skin irritants.

To observe these four requirements it is necessary to have pertinent information regarding both the worker and the job—evaluating the *worker for his abilities* and *disabilities* and evaluating the job for the *physical demands* it imposes and the working conditions under which it is performed.

**Evaluating the Worker.**—Accurate evaluation of the worker requires not only an interview, but also a physical examination—and the examination report should give *as much emphasis* to the worker's abilities as to his disabilities. A diagnosis of a physical disorder is useless if it does not readily convey to the lay placement officer the complete industrial significance of the disorder.

Most existing physical examination reports fall short of these requirements because they are purely *medical* reports expressed in technical language. While they are meaningful to the doctor, they may actually be meaningless to the placement officer. Furthermore, because they are simply an inventory of physical conditions, they incorrectly fix upon the lay placement officer, instead of upon the examining physician, the responsibility of determining the occupational significance of physical disorders. This, too, often results either in overemphasis or in underemphasis of the importance of the disorders, leading to improper placement or unjustifiable rejection of the applicant.

Accurate presentation of useful information resulting from physical examinations requires a functional type of report form which clearly indicates the specific work capabilities and work limitations of the individual. This should be done in the simple English terms which are used in describing the requirements of jobs. This form has the double virtue of presenting to the placement officer a clear, complete picture of the worker's condition and at the same time facilitating the work of the examining physician.

Also important in this connection is a greater knowledge, among lay persons, of anatomy, physiology, and the significance of various disabilities. As in many other occupations and professions where a knowledge of related material is essential to success, so in the case of personnel directors, safety engineers and supervisors, an accurate understanding of the significance of medical terms is invaluable.

**Evaluating the Job.**—Evaluating the job — job analysis or appraisal—is the other essential half of the process of matching workers to suitable jobs. This is a phase of job placement that has been largely neglected. Job appraisal is a simplification of job analysis methods and is designed exclusively for the purpose of employee placement. It is a process by means of which important job factors are rapidly determined and roughly evaluated; it is a systematic, check-list type of survey of each typical job on a record form to determine facts in these four categories: (1) physical demands; (2) working conditions; (3) occupational health hazards; (4) safety hazards.

PHYSICAL DEMANDS.—To understand the physical demands of a job it is necessary to consider both the various abilities and work capacities it requires of the employee and the activities involved in its successful accomplishment. Evaluation should be given to such requirements as agility, strength, exertion, visual skills, hearing, talking, etc., as well as factors such as sitting, standing, walking, running, climbing, crawling, kneeling, squatting, stooping, twisting, lifting, and handling. It is likewise essential that these factors be evaluated according to their degree. What is the duration of an activity? What is the quality or ability needed? What is the severity of the demand upon any worker who is to perform the job? For example, a job involving a considerable amount of stair climbing is unsuitable for workers with heart disease, respiratory diseases, obesity, and with orthopedic disorders of the lower limbs, but a small amount of stair climbing may be tolerated by persons with these disorders.

WORKING CONDITIONS.—Knowledge of conditions under which the job is performed is important because of their possible harmful influence upon certain disabilities. Working conditions include such factors as indoors, outdoors, excessive heat or cold, excessive humidity or dryness, wet working conditions, sudden temperature changes, ventilation and lighting, noise, whether the work is performed alone, near others, or as a member of a crew, shift work, piecework, etc. For example, work in excessive heat is generally unsuitable for persons who have had malaria, or for those with high blood pressure, heart disease, skin disease (particularly acne), the aged (because of their inability to accommodate for loss of salt), and the markedly obese.

Occupational Health Hazards.—This has been the most neglected and least appreciated phase of job placement because it is not well understood by the average layman. Occupational health hazards must be very carefully evaluated, for they have a very insidious effect and are the factors which most frequently cause aggravation of a disability. Such evaluation involves consideration of the following: Air pressure extremes; radiant energy, such as ultraviolet, infrared, radium, and x-rays; silica, asbestos, nuisance dusts; skin irritants; respiratory irritants; systemic poisons and asphyxiants. (Whenever such exposures exist, they should be carefully evaluated, and the services of specially trained industrial hygienists should be used to bring them under control whenever possible. When this is not possible—and even in borderline exposures—it is undesirable to expose individuals having disabilities which are particularly unsuitable.) The reason is that those health hazards usually are prone to affect certain parts or organs of the body more than other parts. Any weakness or pre-existing defect in such parts creates a greater likelihood of damage from exposure which might have no effect on an able-bodied worker. For example, a job might involve exposure to respiratory irritants in such quantities as to be insignificant to a normal person; yet this condition might aggravate the disability of a person who has chronic bronchitis.

Safety Hazards.—Safety—or, rather, fear of excessive accident proneness—has been one of the principal reasons for reluctance to employ handicapped persons. Therefore it is essential that any job appraisal give thorough consideration to the accident hazards involved. This should include: danger of falls from elevators, work on moving surfaces, slipping and tripping hazards, exposure to vehicles or moving objects, falling objects from overhead, foot injuries, eye injuries, cuts and abrasions, burns, mechanical and electrical hazards, fire and explosion hazards. These must be carefully evaluated because of their possible unfavorable relationship to the disability of the handicapped person. For example, a job which involves foot injury hazards is unsuitable for the diabetic because of his susceptibility to gangrene of the feet and slow healing of wounds or union of fractures.

A vital safety factor which demands careful evaluation is responsibility for the safety of others. This refers to occupations in which fellow workers or members of the public would be endangered as a result of imperfect coordination, loss of self-control, or defective functioning of the physical or mental processes of the worker. Examples of persons whose jobs involve responsibility for the safety of others include: airplane pilots, railroad engineers, railroad switch tower operators, bus and streetcar operators, vehicle operators, overhead crane operators.

Using the Information.—Obviously the foregoing information, resulting from job appraisals or job surveys for placement purposes, is of little value unless it is made available to those who hire, select, place, and supervise workers. It is desirable that the employment man have at his fingertips pertinent job information to aid him in hiring workers efficiently. Physicians also will find this information helpful in making physical examinations of workers being considered for specific jobs. Supervisors will often observe that simple revisions of methods will frequently permit the efficient employment of greater numbers of the handicapped. Those concerned with employee welfare will have the advantage of an accurate picture of health and safety conditions which will enable them to take even further steps toward improvement and control.

When both worker and the job are carefully evaluated, the feasibility of hiring handicapped persons will become apparent. It will become obvious that most handicapped persons still "have more ability than disability" and that "few jobs require *all* the abilities of a worker." Only through such a functional, clear, coordinated plan can the long-standing, unwarranted prejudice against handicapped workers be overcome. Only in this way can industry tap a valuable source of manpower, with resulting benefits not only to itself, but also to the economic and social structure of the nation.

Many medical directors devised their own placement program along lines designed to be as simple as possible and devoid of complicated techniques. At Lockheed Aircraft Corporation, Poole[1] used the following method of classifying the applicants:

Group 1: "Unlimited." Acceptable for any type of work.

Group 2: "Limited." If a physical handicap or defect is serious enough to limit a person's working ability, constitute a hazard at work, or be seriously aggravated by some type of work, such a person will be classified as limited and placed in one or more of the following classes, which are intended to describe in lay terms the type of work which he should avoid. The reason for the limitation is a confidential matter and of no practical interest to the supervisor as long as he is aware of the type of work prohibited.

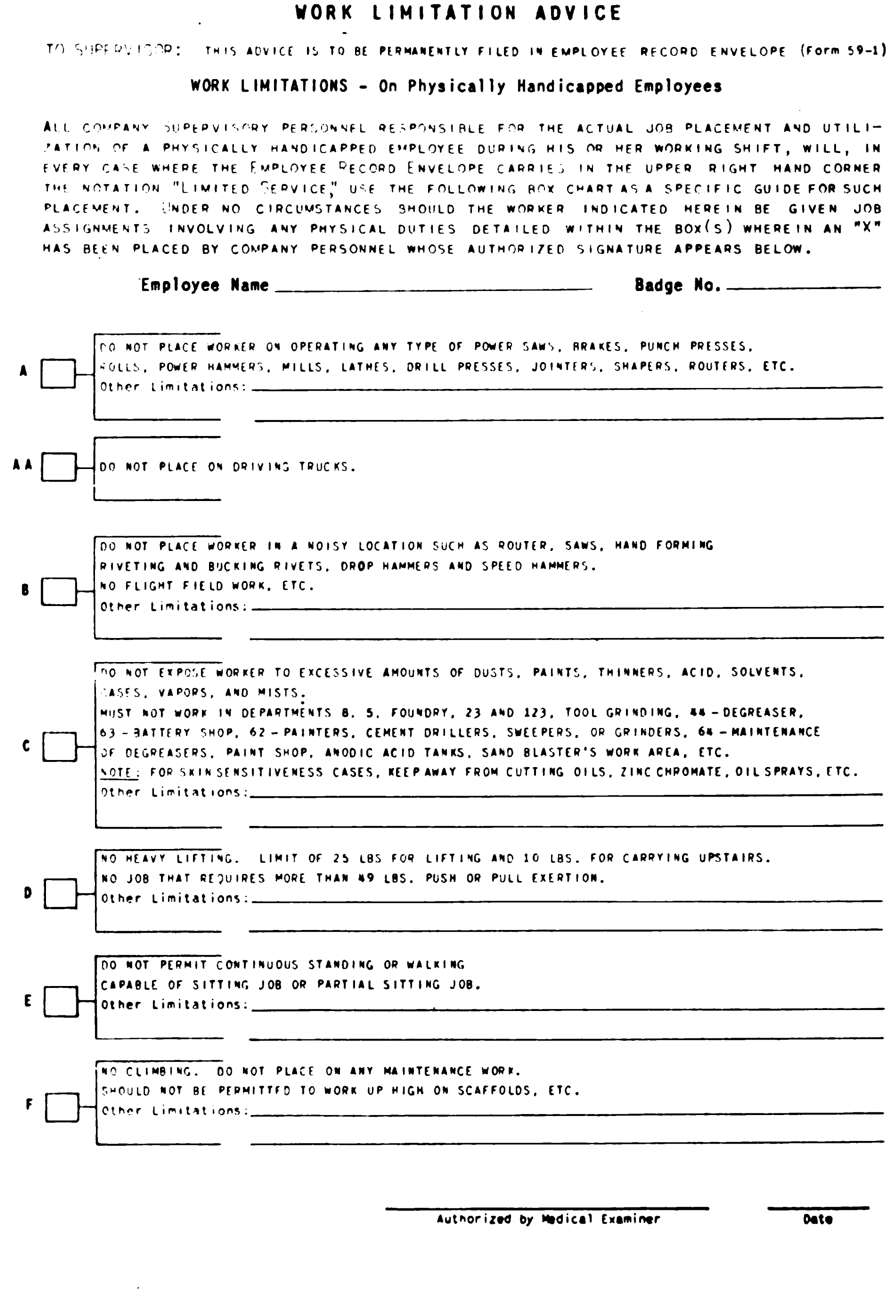
Class I—No hazardous machinery
Class II—No heavy lifting
Class III—Work at ground level only
Class IV—Avoid contact with or exposure to (agent or substance to be specified)
Class V—No excessive walking or standing
Class VI—Miscellaneous (This may be expanded to other classes as needed by the type of industry)

Group 3: "Severe Handicaps" involving special placement problems (as the blind). Transfers are not to be made without approval of the Medical Department.

Group 4: "Temporarily Unable to Work" because of a transitory condition or a correctible defect.

Group 5: "Rejection." Serious physical or mental disease or disability rendering the persons unfit for any work.

At the Northrop Aviation Company, Dr. Harold Dye devised and used the accompanying form for his placement program.



## WORK LIMITATION ADVICE

TO SUPERVISOR: THIS ADVICE IS TO BE PERMANENTLY FILED IN EMPLOYEE RECORD ENVELOPE (Form 59-1).

**WORK LIMITATIONS - On Physically Handicapped Employees**

ALL COMPANY SUPERVISORY PERSONNEL RESPONSIBLE FOR THE ACTUAL JOB PLACEMENT AND UTILIZATION OF A PHYSICALLY HANDICAPPED EMPLOYEE DURING HIS OR HER WORKING SHIFT, WILL, IN EVERY CASE WHERE THE EMPLOYEE RECORD ENVELOPE CARRIES IN THE UPPER RIGHT HAND CORNER THE NOTATION "LIMITED SERVICE," USE THE FOLLOWING BOX CHART AS A SPECIFIC GUIDE FOR SUCH PLACEMENT. UNDER NO CIRCUMSTANCES SHOULD THE WORKER INDICATED HEREIN BE GIVEN JOB ASSIGNMENTS INVOLVING ANY PHYSICAL DUTIES DETAILED WITHIN THE BOX(S) WHEREIN AN "X" HAS BEEN PLACED BY COMPANY PERSONNEL WHOSE AUTHORIZED SIGNATURE APPEARS BELOW.

**Employee Name** ____________________ **Badge No.** ____________

**A** ☐ DO NOT PLACE WORKER ON OPERATING ANY TYPE OF POWER SAWS, BRAKES, PUNCH PRESSES, ROLLS, POWER HAMMERS, MILLS, LATHES, DRILL PRESSES, JOINTERS, SHAPERS, ROUTERS, ETC.
Other Limitations: ____________________

**AA** ☐ DO NOT PLACE ON DRIVING TRUCKS.

**B** ☐ DO NOT PLACE WORKER IN A NOISY LOCATION SUCH AS ROUTER, SAWS, HAND FORMING RIVETING AND BUCKING RIVETS, DROP HAMMERS AND SPEED HAMMERS.
NO FLIGHT FIELD WORK, ETC.
Other Limitations: ____________________

**C** ☐ DO NOT EXPOSE WORKER TO EXCESSIVE AMOUNTS OF DUSTS, PAINTS, THINNERS, ACID, SOLVENTS, GASES, VAPORS, AND MISTS.
MUST NOT WORK IN DEPARTMENTS 8, 5, FOUNDRY, 23 AND 123, TOOL GRINDING, 44 - DEGREASER, 63 - BATTERY SHOP, 62 - PAINTERS, CEMENT DRILLERS, SWEEPERS, OR GRINDERS, 64 - MAINTENANCE OF DEGREASERS, PAINT SHOP, ANODIC ACID TANKS, SAND BLASTER'S WORK AREA, ETC.
NOTE: FOR SKIN SENSITIVENESS CASES, KEEP AWAY FROM CUTTING OILS, ZINC CHROMATE, OIL SPRAYS, ETC.
Other Limitations: ____________________

**D** ☐ NO HEAVY LIFTING. LIMIT OF 25 LBS FOR LIFTING AND 10 LBS. FOR CARRYING UPSTAIRS.
NO JOB THAT REQUIRES MORE THAN 49 LBS. PUSH OR PULL EXERTION.
Other Limitations: ____________________

**E** ☐ DO NOT PERMIT CONTINUOUS STANDING OR WALKING
CAPABLE OF SITTING JOB OR PARTIAL SITTING JOB.
Other Limitations: ____________________

**F** ☐ NO CLIMBING. DO NOT PLACE ON ANY MAINTENANCE WORK.
SHOULD NOT BE PERMITTED TO WORK UP HIGH ON SCAFFOLDS, ETC.
Other Limitations: ____________________

____________________ ________
Authorized by Medical Examiner Date

**Psychiatry and Job Placement.**—It is not possible to establish a completely efficient placement program without the aid of the psychiatrist. Nor is it possible to maintain a completely adequate health program in the plant without the aid of psychiatry. However, too few plants can afford to employ psychiatrists, nor are there enough psychiatrists trained in industrial health methods to supply those plants which would demand such a service. To present the *pros* and *cons* of a psychiatric service in industry would be beyond the scope of this book.

The degree to which psychiatry should be used in the pre-employment examination as well as in the program to maintain the health of those already employed is a moot question. Nor is there general agreement as to who should employ psychiatric methods. Giberson[6] stated that "the industrial physician is the logical medium for the administration of a psychiatric program for the simple reason that industrial psychiatry is at best a supplemental device to general industrial health." It is Poole's[1] opinion that:

> "When workers who are being examined exhibit definite signs of mental derangement, the physician cannot ignore these signs. If the worker does not show obvious signs of derangement, it is my opinion that the examiner should not attempt psychoanalysis, for which he generally is not thoroughly qualified. I believe that the front door to employment should be held open to all but the obviously mentally deranged, but that the physician should keep a watchful eye on the 'back door'—emotional and adjustment problems which arise within the plant. When an employee demonstrates that he is maladjusted to his job or environment, the physician can assist in re-evaluation of the worker. When psychologic factors are responsible, the problem is one for the skilled clinician, who should be fully acquainted with the worker's medical history and industrial environment."

Cameron[7] says that since the most active part of a person's day is spent at work and since many of his major satisfactions and frustrations are met there, psychiatry has begun to find in industry an increasingly important field for work.

Any job analysis which is limited to speed, number of repetitions, degree of coordination, range of movement, amount of lifting, temperature, and humidity is indeed limited. To be a thorough analysis it must include the data as to whether the job can give satisfaction to the worker and the kinds of workers to whom it can give satisfaction. One has still to learn that a process cannot be broken down into separate "operations" on the basis of what is suitable for the machines and assume that the breakdown will also be suitable for the worker. There must still be added to the mechanical and physiologic analysis a psychologic evaluation.

Efforts are now being made to identify those occupations which constitute the greatest psychologic hazard to the worker, or, perhaps, to identify the particular kinds of jobs which are most hazardous for particular kinds of persons.

Certain trades are particularly dangerous for tense persons, who find it hard to relax quickly and adequately between periods of pressure. Occupations which call for sustained utilization of a limited part of their equipment are likely to be harmful. Today it is recognized that if one overloads a man who is excessively set on doing his job well, who cannot be content unless it is done just so, he will begin to manifest tension and anxiety, will lose confidence and concentration, and will eventually have to stay away from work.

It is beginning to be realized that it is hazardous in the industrial medical sense for certain types of personalities to work with certain others. It is more difficult because it requires two separate personality evaluations. The simplest example of this is the situation in which the subordinate is an insecure, unconfident person and the boss is an assertive, sadistic person.

The industrial physician should be well oriented in the field of psychosomatic medicine. The greatest part of the practice of psychiatry cannot be carried out by psychiatrists; it must be carried out by everyone who is engaged in the practice of medicine.

A psychiatrist should examine all new employees. He knows that an overconscientious, precise, and rigid person does not do well in jobs which result in his being pushed beyond his usual tempo of work. By recognizing the existence of such personality traits during the medical examination, he can assist, through recommendations as to placement, and thus serve to prevent subsequent breakdowns. Adequate selection and placement will go far not only in increasing effectiveness but also in cutting down the widespread mental ill health which exists.

Industrial psychiatric research should also concern itself with the investigation of psychologically hazardous occupations and the study of situations in which an unusually high degree of maladjustment and mental ill health develops. Every month men leave industry permanently damaged by exposure to too much tension for too long. Most commonly these men are precisely the ones whom industry most values—men who are conscientious, precise in their work, and more than willing to take responsibility. Industry has a major obligation to see that means are worked out to protect these workers against the dangers to which their very virtues expose them.

Psychiatry is an emotional first-aid station in industry, according to Lott.[8] He believes that a phychiatrist on the company's medical department staff can be of definite assistance. If possible he should take regular duty hours. Even once a week may be helpful. He is then available to workers through routine nonalarming channels. He can also assist and interest the various staff members in caring for the various degrees of emotional sickness encountered among employees. Psychiatric interview techniques and findings may be needed in questions of medical authorization for job transfers, leaves, and releases, when handicapping physical findings are not sufficiently prominent or are entirely absent. Minor neurotic conversion symptoms often mask causative home and social problems, a knowledge of which would be useful to the personnel department in making their decisions. Pre-employment medical-personality evaluations, in the hands of an expert personnel staff, may be helpful in efficiently utilizing available manpower. The emotionally or physically handicapped returned servicemen are especially worthy of such efforts. The problems incident to postwar conversion will only multiply the need for the most adequate personnel policies and practical application of them.

In addition to the general application of psychiatry in industry, there is an increasing demand for trained interviewers and counsellors. It is believed that much time and money could be saved by weeding out the mentally unfit or the maladjusted worker at the door of the employment office. It is held that these unfit individuals would never have to be examined and refused employment if the interviewer were trained in proper selection techniques. Here again one is dealing with a controversial question and certainly the right to pass the applicant for further examination and employment or deny the applicant this privilege requires a most thoroughly trained and efficient person.

On the other hand, trained counsellors within the plant have a definite place in any industry employing fairly large groups of workers. Many individuals with attitudes of frustration or resentment, or those who are fatigued or fed up with the job, can be aided by a wise counsellor. It has been estimated that about 20 per cent of workers are unable to bear efficiently the stresses of industrial friction. For these, as well as for the mentally unstable applicant for employment, some measure of a psychiatric program is needed, and its eventual inclusion in industrial medicine is inevitable.

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# APPENDIX

## THE CHEMICALS IN COMMON TRADE-NAME PRODUCTS

Frequently the industrial physician finds it necessary to know the chemical composition of a given product, the nature of which is not revealed by the name or the label. Since such information is not readily accessible, the following list is presented. Many products have been omitted because the nature of their chemical composition was not available to the author. Also others were omitted because their use was confined to a highly specialized industry and therefore not of general interest.

The information presented here has been gleaned from such sources as chemical dictionaries, manufacturer's catalogues, etc. Special acknowledgment is due to *Handbook of Material Trade Names* by O. T. Zimmerman and Irvin Lavine, Industrial Research Service, Dover, N. H., 1946, and to Allen D. Brandt, W. J. McConnell, and R. H. Flinn, *Public Health Reports*, vol. 61, no. 5, pp. 132-143, reprint no. 2691.

**A-10.** A rubber accelerator. Condensation product of aniline and formaldehyde, probably a mixture of methylene dianilide and formaldehyde aniline.
MONSANTO CHEMICAL CO.

**A-11.** A rubber accelerator consisting of a specially-processed formaldehyde derivative of a condensation product of aniline and acetaldehyde.
MONSANTO CHEMICAL CO.

**Accelerator 552** (*Also known as* PIP-PIP). Piperidinium pentamethylenedithiocarbamate. A fluffy, cream-colored powder. *Use:* To accelerate and improve the vulcanization of rubber and rubber-latex compounds.
DUPONT CO.

**Accelerator No. 8.** Formaldehyde-para-toluidine. A white powder with a grayish-yellow cast and an aromatic odor. Sp.gr. 1.11; soluble in acetone. *Use:* As a rubber vulcanizer. DUPONT CO.

**Acrol.** 2,4-Diaminophenol dihydrochloride. A photographic developing chemical in the form of white crystals; very soluble in water; slightly soluble in alcohol. EASTMAN KODAK CO.

**Aerocarb.** A mixture of sodium, potassium, and barium salts. *Use:* As a molten bath for the case hardening and heat treatment of steel.
AMERICAN CYANAMID & CHEMICAL CORP.

**Aerocase.** A mixture of sodium and calcium chlorides and calcium cyanide. *Use:* As a molten bath for the case hardening and heat treatment of steels.
AMERICAN CYANAMID & CHEMICAL CORP.

**AgeRite Powder.** Phenyl-beta-naphthylamine. A rubber antioxidant consisting of a gray, free-pouring powder with a faint odor. R. T. VANDERBILT CO.

**AgeRite Resin.** Aldol-alpha-naphthylamine. A rubber antioxidant consisting of a cherry-red resin with a faint odor. R. T. VANDERBILT CO.

**Albatex BD.** Sodium m-nitrobenzenesulfonate. A water-soluble, white powder that counteracts the action of reducing agents, and thus finds special use protection of the dye colors in textile dyeing, printing, and finishing. Ciba Co.

**Alberene Sawdust.** Soapstone dust. An impure, natural, hydrous magnesium silicate. A soft powder having a soapy or greasy feel. *Use:* As a filler in paper, rubber, soap, plastics, etc. Alberene Stone Corp. of Va.

**Albone.** Hydrogen peroxide. A colorless, slightly pungent liquid which liberates oxygen in reaction. *Uses:* As a bleaching agent for textiles; as an oxidizing agent for vat dyeing; as a source of oxygen. DuPont Co.

**Alcotone No. 1.** A proprietary solvent consisting of a mixture of acetone, a smaller quantity of denatured ethyl alcohol, and small amounts of esters, hydrocarbons, and wood alcohol. *Uses:* As a solvent for Celluloid, nitrocellulose, cellulose acetate, oils, greases, and many gums and resins. Commercial Solvents Corp.

**Alkanol S.** Sodium tetrahydronaphthalene sulfonate. A dispersing agent in the form of a light cream-colored powder, readily soluble in water. *Uses:* In textile printing; in soap to increase lathering power; and in the preparation of stable emulsions of carnauba wax. DuPont Co.

**Alorco Cryolite.** Sodium fluo-aluminate. A finely-divided, bulky, white powder, with a fluorine content of 46.2%. *Use:* To control chewing insects on most vegetable crops. Aluminum Ore Co.

**Alsilox.** A reaction product of lead oxide and silica. It is used for the preparation of ceramic glazes. Supplied in granular, lump, and powdered forms. Eagle-Picher Lead Co.

**Alvar.** A polyvinyl acetal resin, slightly yellow in color, available in several grades. A viscous liquid; soluble in lacquer solvents and aromatic hydrocarbons; insoluble in aliphatic hydrocarbons. *Uses:* In lacquers, adhesives, coatings, phonograph records; and in the form of sheets, rods, and tubes. Shawinigan Products Corp.

**Amidol.** 2,4-Diaminophenol dihydrochloride. White to grayish-white crystals; slightly soluble in alcohol; very soluble in water. *Use:* As a photographic developing chemical. Associated Chemists, Inc.

**Amyloform.** A formaldehyde-carbohydrate condensation product. It is an extremely finely divided, soft, white powder with a large specific surface. *Uses:* As a filler and extender for cements, puttys, adhesives, enamels, paints, explosives, insecticides, preservatives, and molded plastics; and as a reinforcing agent for natural and synthetic rubber. Perkins Glue Co.

**Armaline T.S.P. "N".** Commercial trisodium nitrophosphate. $4Na_3PO_4 \cdot NaNO_3 \cdot 44H_2O$. Mol.wt. 1533.77. *Uses:* In packaged water softeners, cleaners, and washing compounds; and as a soap assistant in clothes washing. A. R. Maas Chemical Co.

**Armasol.** A mixture of anhydrous tetrasodium pyrophosphate ($Na_4P_2O_7$), and trisodium nitrophosphate ($4Na_3PO_4 \cdot NaNO_3 \cdot 44H_2O$). *Uses:* As a detergent and water softener in dishwashing machines; and as a general household cleaner. A. R. Maas Chemical Co.

**Armstrong's Acid.** A technical grade of naphthalene-1,5-disulfonic acid. $C_{10}H_6(SO_3H)_2$. Mol.wt. 228.28. A white, crystalline solid, soluble in water. *Use:* As a dyestuff intermediate.

**Artic.** Methyl chloride refrigerant. $CH_3Cl$. *Uses:* As a refrigerant; as a methylating agent in organic synthesis; and as a fluid for thermometric and thermostatic devices. DuPont Co.

**Banana Oil.** *Also known as* Pear Oil. Amyl acetate. $CH_3COOC_5H_{11}$. *Uses:* In the manufacture of gun cotton; and in fruit flavors, food preparations, cosmetics, photo- and synthetic rubber; and to reduce surface tack in reclaim stocks.

**Baroid.** Pulverized barytes, $BaSO_4$, designed for use as a weighting material for oil-well drilling muds to increase the unit weight and thereby control formation pressures and prevent caving. Baroid Sales Div. National Lead Co.

**Beetle Mort.** A combined insecticidal and fungicidal dust containing 21% arsenic oxide and 5% metallic copper. It is used as a dust, or is dispersed in water and used as a spray. John Lucas & Co.

**Benclor-3.** A chlorined benzene used to control and kill aquatic weeds, algae, slime, etc., in ponds, streams, and drainage ditches. It is a clear liquid with a pungent odor; sp.gr. 1.45; practically insoluble in water. It is applied by forcing it into the water through fine nozzles in order to produce a milky emulsion. Available in three grades: Regular, Special, and C. The regular grade is especially adapted for static water, and the other two for running water. Cloroben Corp.

**Blacosolv.** Trichloroethylene. $C_2HCl_3$. A colorless, stable, low-boiling, heavy, mobile, toxic, nonflammable, nonexplosive liquid. It does not attack the common metals even in the presence of moisture. *Uses:* As a metal-degreasing solvent; as a solvent for gums, resins, waxes, fats, etc.; as a dry-cleaning solvent; and in paint and varnish removers, etc. G. S. Blakeslee & Co.

**Bordow.** A light-blue, finely divided copper fungicide that is applied as a dust or as a wet spray. *Uses:* To control blights and fungous growths on fruits, shrubs, and vegetables. Dow Chemical Co.

**Bore-Tox.** A combination of paradichlorobenzene and a 98% miscible mineral oil. Used in a concentration of 1 gal. of Bore-Tox to 7 gal. of water to kill peach-tree borers. Miller Chemical & Fertilizer Corp.

**Broenner's Acid.** A technical grade of 2-naphthylamine-6-sulfonic acid. $NH_2C_{10}H_6SO_3H \cdot H_2O$. Mol.wt. 241.26. Colorless, needlelike crystals; soluble in boiling water. *Use:* As a dyestuff intermediate.

**Bug Geta Pellets.** Metaldehyde with an arsenical bait, in pellet form. For use against slugs, snails, cut worms, and grasshoppers. California Spray Chemical Co.

**Butanol.** Normal butyl alcohol. Commercial Solvents Corp.

**Butyl Cellosolve.** $C_4H_9OCH_2CH_2OH$. A colorless, odorless, liquid glycol ether, completely miscible with water. *Use:* As a solvent. Carbide & Carbon Chemicals Corp.

**Buzz.** A household insecticidal spray consisting of 1.04% DDT and 3.14% Thanite in a nonstaining, nonirritating petroleum solvent. This spray gives 100% knockdown in 10 minutes and 100% kill in 24 hours. Because of the low DDT content, the residual effects are slight; however, insecticidal sprays of this type are less toxic than those with higher DDT contents. *Use:* Principally for the rapid elimination of such common household insects as flies and mosquitoes. Chemical Research Products Co.

**Carbo.** A group of clay-bonded silicon carbide refractories containing from 30 to 50% SiC. Carborundum Co.

**Carbolineum.** An anthracene oil used for killing chicken mites, as a wood preservative, and for the control of termites. It may be used in concentrated form or diluted with kerosene. CARBOLINEUM WOOD PRESERVING CO.

**Carbon Met.** A volatile, water-white, methylated hydrocarbon solvent for metal cleaning. It is noncorrosive, nonflammable and nonexplosive; and dissolves readily in gums, oxidized oils, burnishing compounds, etc. CURRAN CORP.

**Carbowax.** A group of nonvolatile, solid polyethylene glycols, soluble in both water and aromatic hydrocarbons. They resemble natural waxes in appearance and texture but are soluble in a much wider range of solvents. Their aqueous solutions possess binding and surface-active properties.

**Carboxide.** A colorless gas consisting of a mixture of 10% ethylene oxide ($CH_2CH_2O$) and 90% carbon dioxide ($CO_2$). *Uses:* As an insecticide and fumigant for moths and vermin; and for controlling mold and bacteria.
CARBIDE & CARBON CHEMICALS CORP.

**Cecolene No. 1.** *Same as* TRI-CLENE. Trichloroethylene. $C_2HCl_3$. A colorless, stable, low-boiling, heavy, mobile, toxic, nonflammable, nonexplosive liquid with a chloroform-like odor. Miscible with all common organic solvents. *Use:* As a metal-degreasing solvent. DUPONT CO.

**Cecolene No. 2.** *Same as* PER-CLENE. Tetrachloroethylene. $C_2Cl_4$. A colorless, stable, nonflammable liquid, with an etherlike odor. Miscible with most organic solvents; insoluble in water. *Use:* As a metal-degreasing solvent.
DUPONT CO.

**Cellosolve.** Ethylene glycol monoethyl ether. $C_2H_5OCH_2CH_2OH$. Completely soluble in water; miscible with hydrocarbons. *Uses:* As a solvent for nitrocellulose, resins, lacquers, etc.; in soluble soaps, insecticides, dry-cleaning soaps, etc. CARBIDE & CARBON CHEMICALS CORP.

**Ceramitalc.** Natural hydrous magnesium silicate (talc). $H_2Mg_3(SiO_3)_4$. A fine white powder, 99% of which passes through a 325-mesh screen. *Uses:* As a filler for paints, plastics, plaster, roof cements, paper, etc.; in pharmaceuticals; in textile finishing, etc. INTERNATIONAL PULP CO.

**Chem-D.** A residual type insect spray consisting of 5% DDT (dichlorodiphenyltrichloroethane) in a petroleum solvent. It is very effective against flies, mosquitoes, bedbugs, roaches, silverfish, fleas, lice, moths, ticks, mites, ants, etc. It is best applied with an atomizing sprayer to all surfaces with which insects come in contact—walls, ceilings, screens of homes, stores and factories; interiors of barns, poultry houses, etc.; manure piles and other infested areas. A single application leaves a deposit of DDT that retains its killing power for long periods —a few weeks to a few months in open places exposed to sunlight, and up to several months in closets and other enclosed places.
CHEMICAL RESEARCH PRODUCTS CO.

**Chemigum.** A butadiene-acrylonitrile polymer synthetic rubber. It resembles brown, crepe natural rubber, and possesses a distinctive odor. Tensile strength (vulcanized), 4425 lb./sq.in.; elongation, 630%; slightly superior to natural rubber in resistance to abrasion; very much less soluble in oils and solvents than natural rubber. Available in three grades of hardness: I, II, and III. *Use:* As a substitute for natural rubber, especially in contact with oil, grease, and gasoline. GOODYEAR TIRE & RUBBER CO.

**Chlorasol.** A chlorinated solvent consisting of 75% ethylene dichloride by volume and 25% carbon tetrachloride. Sp.gr. 1.34. It possesses good solvent power and is nonflammable under ordinary conditions. *Uses:* As a fumigant for the control of moths and other insects; for fumigating flourmill machinery; and for fumigating seeds and bulbs of all kinds. CHEMICAL SUPPLY CO.

**Chlorcosane.** A chlorinated paraffin with a combined chlorine content of 27-30%. It is a light yellow, odorless, and tasteless oil that possesses solvent, fire-resisting and insulating properties. Sp.gr. 1.00-1.07.
MONSANTO CHEMICAL CO.

**Chlorowax.** A white, pulverized, chlorinated, resinous paraffin. Sp.gr. 1.64; m.p. 90° C.; insoluble in water; soluble in hydrocarbons, ketones, esters, nitroparaffins, and chlorinated hydrocarbons; compatible with VINYLITE, chlorinated rubber, synthetic rubbers, synthetic resins, natural waxes and gums; does not oxidize, polymerize, or condense; decomposes at 135° C. with evolution of hydrogen chloride; in presence of calcium carbonate, decomposition is controlled and reaction products—$CO_2$ and $CaCl_2$—act as flame retardants. *Uses:* In flame-retarding paints; for water-proofing paper; in electric-cable coating compositions; in lacquers; in adhesives; in textile flame-proofing compositions.
DIAMOND ALKALI CO.

**Chlorpropane Liquid 170.** A clear, colorless, nonflammable liquid with a mild, characteristic odor. It consists of 40-50% heptachlorpropane, 0-5% pentachlorpropane, and 0-5% octachlorpropane. Sp.gr. 1.70-1.75; b.p. range, 160-260° C., (760 mm.). Insoluble in water but infinitely soluble in alcohol, ether, and most chlorinated solvents; compatible with various synthetic resins and rubbers. *Uses:* As a plasticizer; solvent, etc. HOOKER ELECTROCHEMICAL CO.

**Chlorpropane Wax 130.** A highly chlorinated propane derivative in the form of a tough, white, crystalline wax with a mild camphorlike odor. It consists of 85% octochlorpropane and 15% heptochlorpropane. Insoluble in water, but soluble in alcohol, ether, and most chlorinated solvents; and compatible with various synthetic resins and rubbers. *Uses:* As a plasticizer; as a dielectric wax; in pyrotechnic compositions; and as a chemically resistant lubricant.
HOOKER ELECTROCHEMICAL CO.

**Clor-O-Tol.** A concentrated hypochlorite bleach solution for laundry use. It prevents mildew and removes stains. CLARKSON CHEMICAL CO.

**Clora Kleen.** A chlorinated cleaning powder containing 5% calcium hypochlorite (70% available chlorine) and 75% cleaning compounds. It is soluble in hot or cold water. *Use:* For cleaning glassware, dishes, bottles, and dairy equipment. CHEMICAL MFG. & DISTRIBUTING CO.

**Cloroben.** An amber-colored, nonflammable liquid, composed of chlorinated hydrocarbons, that forms a milky emulsion when mixed with water. It is chemically stable, and does not liberate chlorine or form phenols. When added to sewage, it hinders putrefaction, slime, and hydrogen sulfide formation; and also destroys the larvae of flies, mosquitoes, etc. *Use:* To reduce odors arising from sewers, septic tanks, sewage-settling beds, etc. CLOROBEN CORP.

**Cloros (12¾%) Dry Bordo.** A copper-containing fungicidal dust containing 12¾% metallic copper. It should not be used on stone fruits.
JOHN LUCAS & CO.

**Clorox.** A sodium hypochlorite solution for bleaching, deodorizing, and disinfecting purposes. It contains 5.25% by weight of NaOCl.
CLOROX CHEMICAL CO.

**Colonial Spirits.** Pure methyl alcohol. $CH_3OH$. Mol.wt. 32.03. A clear, colorless, volatile, poisonous, flammable liquid. Sp.gr. 0.792; b.p. 64.7° C.; m.p. —97.8° C. *Uses:* As a solvent; an intermediate in the synthesis of other organic compounds; a fuel; a denaturant for ethyl alcohol; an automotive antifreeze.

**Colorex Stripper.** A dark-colored liquid for stripping colors from dyed fabrics. It is a powerful reducing agent and is decomposed by alkaline materials such as caustic soda. Available in 2 concentrations: No. 20 analyzes 20.0% titanium trichloride, 0-5% titanium tetrachloride, 15.6% zinc chloride, 7-9% hydrochloric acid, and 50-57% water and stabilizer; No. 23 analyzes 23% titanium trichloride, 0-5% titanium tetrachloride, 18.5% zinc chloride, 7-9% hydrochloric acid, and 44 to 51% water and stabilizer. NIAGARA DIV. STAUFFER CHEMICAL CO.

**Creotex.** A liquid creosote concentrate that is used as a wood preservative. For use, one gallon of the concentrate is added to 10 gallons of old crank-case oil or crude petroleum. INDUSTRIAL MATERIALS CO.

**Cresolave.** A cresylic acid disinfectant for use in vehicles, yards, and pens where livestock are handled. CHEMICAL SUPPLY CO.

**Cresolene.** A coal-tar disinfectant made from distilled tar acids. It readily forms milky emulsions with water. CLIFTON CHEMICAL CO.

**Cyanegg.** A white, fused solid in the form of 1-oz. eggs, containing a minimum of 96% sodium cyanide ($NaCN$). *Uses:* For heat-treating steel; in electroplating; in the cleaning of metal, including neutralization after pickling; in fumigation; for the manufacture of indigo, dye intermediates, and other chemical compounds; in the elimination of metal impurities from industrially used water; and in the extraction of gold from ores. DUPONT CO.

**Cyanobrite.** A red, powdered mercury salt that is soluble in acids and alkalies. *Use:* As the mercury-carrying factor for R & H DUOZINC plating solutions. DUPONT CO.

**Cyanogas.** Calcium cyanide dust and flakes used for rodent and insect control. AMERICAN CYANAMID & CHEMICAL CORP.

**DDT.** Dichloro-diphenyl-trichlorethane. $CCl_3CH(C_6H_4Cl)_2$. Mol.wt. 354.50. A white, stable, practically odorless, crystalline powder possessing remarkable insecticidal and lousicidal properties. It is insoluble in water, but is readily soluble in alcohol, acetone, cyclohexanone, benzol, toluol, xylol, and a wide range of petroleum oils and solvents. Its degree of toxicity varies with the form in which it is used: when taken by mouth in small amounts, it is moderately toxic to warm-blooded animals; when used in dust form, it is not adsorbed and is nonirritating to the skin; but in solution, it is definitely toxic, especially if the vehicle is readily absorbed by the skin.

**Dactin.** Dichloro dimethylhydantoin. $(CH_3)_2C(NClCO)_2$. Mol.wt. 197.3. A white, crystalline active-chlorine compound with a minimum of 66% available chlorine. It can be cast and melted into any desired shape; is soluble in organic solvents, particularly chlorinated hydrocarbons; and is slightly soluble in water. *Uses:* As a general chlorinating and oxidizing agent where solubility in organic solvents and an inactive residue are important; as a dye developer in textiles, etc. DUPONT CO.

**Di-Chlor-Mulsion.** A 90% emulsion of ethylene dichloride. It is diluted and poured around the base of trees to control peach-tree borers. TAYLOR CHEMICAL WORKS

**Dicalite.** Diatomaceous silica. $SiO_2$. Very finely divided diatomaceous particles, acicular and elongated in shape. DICALITE CO.

**Dicarbo.** Silicon Carbide. $SiC$. A highly refractory, extremely hard, crystalline product of the electric furnace. *Uses:* In the form of grains and powders for cutting, polishing, and grinding; for the manufacture of grinding wheels, stones, hones, dental points, etc.; for the manufacture of tiles, bricks, blocks, etc. JAMES H. RHODES & CO.

**Diethyl Carbitol.** $O(C_2H_4OC_2H_5)_2$. Mol.wt. 162.23. A colorless liquid ether, miscible with water. B.p. 187.9° C.; sp.gr. 0.9094. *Use:* As a solvent.
CARBIDE & CARBON CHEMICALS CORP.

**Diethyl Cellosolve.** $C_2H_5OC_2H_4OC_2H_5$. Mol.wt. 118.17. A colorless liquid ether, slightly soluble in water. B.p. 121.4° C.; sp.gr. 0.8424. *Use:* As a general solvent.
CARBIDE & CARBON CHEMICALS CORP.

**Dithane.** Diethylene-sodium-misdithiocarbamate. A synthetic insecticide and fungicide. It is sprayed on the ground where it is absorbed by food-producing plants, rendering them immune to insect and fungi destruction. It has been found especially helpful in increasing the yield of potato crops. ROHM & HAAS CO.

**Dow Weed Killer No. 24.** A dry, white, powdered herbicide containing 70% by weight of 2,4-dichlorophenoxyacetic acid (2,4-D) and 30% of dispersing agents. It is used in the form of a spray (about 1⅛ oz. 2,4-D to 10 gal. water) to kill dandelions, plantains, etc., in lawns without damage to the common grasses—Kentucky bluegrass, redtop, fescue, buffalo grass, etc. It is also effective on poison ivy and numerous other weeds. DOW CHEMICAL CO.

**Dung Salt.** Sodium arsenate. $Na_2HAsO_4 \cdot 12H_2O$. Mol.wt. 402.10. A colorless, crystalline solid; sp.gr. 1.72; m.p. 28° C.; soluble in water; slightly soluble in alcohol. *Use:* In the manufacture of insecticide and germicidal compounds.

**Duponol.** A series of higher aliphatic alcohol sulfates and their derivatives used as wetting, penetrating, and dispersing agents, and detergents. Several of the DUPONOL products are identical with those offered under the name GARDINOL. A number of the Duponols are described below. DUPONT CO.

**DuPont Potato Dust.** A combination insecticide and fungicide dust containing calcium arsenate insecticide and COPPER-A COMPOUND fungicide. *Use:* For the control of Colorado potato beetles, flea and blister beetles, hornworms, leaf hoppers, early and late blight, and other leaf-spot diseases. It may also be used on tomatoes, eggplants, and peppers, but fruits so treated must be washed before use. DUPONT CO.

**Dutch Liquid.** Ethylene dichloride. $ClCH_2CH_2Cl$. Mol.wt. 98.97. A colorless liquid of characteristic odor. Sp.gr. 1.258; m.p.—35.3° C., b.p. 83.7° C.; very slightly soluble in water; soluble in alcohol and ether. *Uses:* As a cleaning fluid; as a fumigant; and as an intermediate in the manufacture of chemicals.

**En-Dow-Weed.** A mixture of 2,4-dichlorophenoxyacetic acid (2,4-D) and a sodium salt that aids in dispersing the 2,4-D in water. *Use:* In water solution as a weed-killing spray. DOW CHEMICAL CO.

**Erustocide.** An acid fluorine salt type laundry sour especially suitable for colored fabrics. It acts as a germicide, brightens colors, and imparts luster and softness to flat work. PENNSYLVANIA SALT MFG. CO.

**Esotoo.** Commercial liquid sulfur dioxide. $SO_2$. Mol.wt. 64.06. A colorless liquid with a characteristic, pungent odor. B.p. 14.0° F.; critical temp. 314.8° F.; critical pressure, 1141.5 lb./sq.in. *Uses:* As a reagent in various chemical reactions; in the packing of fruits; as an antichlor in textile dyeing; and in the refining of beet sugar. VIRGINIA SMELTING CO.

**Ethanol.** Ethyl alcohol. $C_2H_5OH$. Mol.wt. 46.07. A clear, volatile liquid with a characteristic vinous odor and pungent taste. Sp.gr. 0.789 (20° C.); b.p. 78.4° C.; infinitely soluble in water, chloroform, and ether. *Uses:* As a solvent; as an intermediate in organic synthesis; as an automotive antifreeze; in pharmaceuticals, etc.

**Ethylbutyl Cellosolve.** $(C_2H_5)_2CHCH_2OCH_2CH_2OH$. Mol.wt. 146.23. A colorless liquid glycol ether slightly soluble in water. B.p. 196.8° C.; sp.gr. 0.8952. *Use:* As a general solvent. CARBIDE & CARBON CHEMICALS CORP.

**Ethylhexyl Cellosolve.** $C_4H_9CH(C_2H_5)CH_2OC_2H_4OH$. Mol.wt. 174.28. A colorless, liquid glycol ether, practically insoluble in water. B.p. 228.3; sp.gr. 0.8859. *Use:* As a solvent. Carbide & Carbon Chemicals Corp.

**Freon.** A group of fluorochloromethanes and ethanes possessing thermal, physiologic, and chemical properties which make them very desirable for use as refrigerants in domestic and commercial appliances. They are stable, colorless, nontoxic, nonirritating, nonflammable, noncorrosive liquids with an ethereal odor. The properties of several of the more important of these compounds are as follows:

| TRADE NAME | CHEMICAL NAME |
|---|---|
| Freon- 11 | Trichloromonofluoromethane |
| Freon- 12 | Dichlorodifluoromethane |
| Freon- 21 | Dichloromonofluoromethane |
| Freon-114 | Dichlorotetrafluoroethane |

Kinetic Chemicals, Inc.

**Fuller's Earth.** A porous, adsorptive clay consisting of hydrated aluminum and magnesium silicates. *Uses:* As a filler for rubber; as a decolorizing agent for oils, fats, greases, etc.; as a carrier for pigments; and as a catalyst in petroleum refining.

**Fusel Oil.** A clear, volatile, poisonous, oily liquid containing principally isomeric amyl alcohols, along with acids, esters, and aldehydes. Sp.gr. of the crude oil, 0.832; distillation range, 110-138° C.; soluble in water, alcohol, ether, and gasoline. *Uses:* In chemical synthesis; as a flavoring material; and as a solvent for fats and oils.

**G.B.S. Soda.** Sodium bisulfate ($NaHSO_4$) in the form of white, opaque or translucent, globular-shaped pellets. Soluble in water with strong acid reaction; decomposes in alcohol. *Use:* As an ingredient in toilet-bowl cleaners, and as a substitute for sulfuric acid. DuPont Co.

**Grasselli Standard.** A lead arsenate insecticide in the form of a white or colored poisonous powder containing at least 96% lead arsenate and not over 4% inert ingredients. *Uses:* As an insecticide for spraying or dusting vegetation such as vegetables, fruits, and shrubs, for the destruction of insect pests such as the Japanese beetle, coddling moth, potato beetle, and other leaf-destroying insects. DuPont Co.

**Hasting's Naptha.** Methyl alcohol. $CH_3OH$. Mol.wt. 32.03. A clear, colorless, volatile, poisonous, flammable liquid. Sp.gr. 0.792; b.p. 64.7° C.; m.p. —97.8° C. *Uses:* As a solvent; an intermediate in the synthesis of other organic compounds; a fuel; a denaturant for ethyl alcohol; an automotive antifreeze.

**Hexalin Solvent.** Cyclohexanol. $C_6H_{11}OH$. Mol.wt. 100.16. A water-white, hygroscopic, oily liquid with a camphorlike odor. Sp.gr. 0.937; m.p. 23° C.; b.p. 160.9° C.; flash point, 68° C.; sparingly soluble in water; miscible with most organic solvents and oils. *Use:* As a solvent, homogenizer, and stabilizer in the paint and varnish, soap, dry-cleaning, and textile industries. DuPont Co.

**Hi-Cyan.** Sodium cyanide. NaCN. Mol.wt. 49.01. A white, crystalline, water-soluble solid. *Uses:* In electroplating gold and silver, and in the extraction of gold from ores. General Chemical Co.

**Hold-Tite.** A naphthalene acetic acid spray. It is a solution of a synthetic hormone that is used to prevent the premature dropping of fruit from trees. For use, it is diluted in the ratio of 1 pint per 100 gallons of water.

John Lucas & Co.

**Hy-Chlor.** Solid calcium hypochlorite. $Ca(OCl)_2{\cdot}4H_2O$. Mol.wt. 215.06. A disinfectant and bleaching agent with an available chlorine content of not less than 70% by weight. F. M. SPECKMAN CO.

**Hypo.** Sodium thiosulfate. $Na_2S_2O_3{\cdot}5H_2O$. Mol.wt. 248.19. White, translucent crystals; sp.gr. 1.729; m.p. 48° C.; soluble in water and ammonia, very slightly soluble in alcohol. *Uses:* As a photographic fixative; in tanning; in the extraction of silver from its ores; and as a disinfectant, mordant, chemical reagent, etc.

**Hyporice.** Rice-sized crystals of sodium thiosulfate. $Na_2S_2O_3{\cdot}5H_2O$. Mol.wt. 248.19. Sp.gr. 1.729; m.p. 48° C.; soluble in water; insoluble in alcohol. *Use:* In photographic work, as a fixing agent to dissolve unchanged silver salts from exposed negatives. MALLINCKRODT CHEMICAL WORKS.

**Kil-Sect.** Air-floated arsenate of lead dust containing not less than 30% arsenic oxide and less than 1% water-soluble material. *Use:* As an insecticide dust. JOHN LUCAS & CO.

**Kill-Tone Powder.** A combined insecticide and fungicide containing 10½% arsenic oxide and 12¾% combined copper. It may be used as a dust, or as a spray in concentrations of 3 to 4 lb. to 50 gallons of water. JOHN LUCAS & CO.

**Lactol Spirits (Amsco).** A petroleum naphtha with a distillation range of 200-265° F. and a sp. gr. of 0.7313. *Uses:* In lacquers, rubber, cements, and rubber tires; for the extraction of rosin, castor oil, and fats; in textile proofing and printing; for leather degreasing, etc. AMERICAN MINERAL SPIRITS CO.

**Lethane B-71.** A thiocyanate insecticide. A powerful insect killer used to control aphids, leafhoppers, and other insect pests. Available as a dry concentrate which requires only simple extension with proper carriers for the manufacture of the finished dust. ROHM & HAAS CO.

**Lignasan.** A white powder fungicide containing ethyl mercuric chloride. *Uses:* To control fungus blue stain (sap stain) in freshly sawed lumber; to retard the formation of slime in paper-mill machinery; and to prevent the deterioration of stored groundwood pulp due to mold and bacteria. DUPONT CO.

**Liquer Chloro-Hyde.** A solution containing chlorine and formaldehyde furnished in several odors. *Use:* As a disinfectant spray for public buildings, halls, etc. CLARKSON CHEMICAL CO.

**M-22.** A dust of 80% sulfur and 20% of a lead compound. *Use:* For summer dusting of apple trees to control certain fungus diseases. MILLER CHEMICAL & FERTILIZER CO.

**Magnesol.** A highly adsorptive synthetic hydrous magnesium silicate, available in a number of modifications for specific applications. *Uses:* As a special purpose filler compound; as a deodorant powder or ingredient; as an anticaking additive in resinous molding powders; for decolorizing crude and reclaimed lube oils; for conditioning dry-cleaning solvents; and for removing colors, odors, and acids from nonaqueous media. WESTVACO CHLORINE PRODUCTS CORP.

**Merclor.** Sodium hypochlorite (NaOCl) in the form of a solution containing 12% available chlorine. *Uses:* As an oxidizing agent for the bleaching of vegetable fibers, and as a chlorinating agent for water. MONSANTO CHEMICAL CO.

**Merko.** A mercury-containing product for treating seed corn to prevent seedling rot and thus permit earlier planting. PITTSBURGH PLATE GLASS CO.

**Methanol.** *Also known as* WOOD ALCOHOL. $CH_3OH$. Mol.wt. 32.04. A clear, volatile, poisonous, flammable liquid. Sp.gr. 0.792; m.p. —97.8° C.; b.p. 66.7° C.

*Uses:* As a solvent; as an intermediate in the synthesis of other organic compounds; as a fuel; as a denaturant for ethyl alcohol; as an automotive antifreeze, etc.

**Methyl Carbitol Acetate.** Diethylene glycol monomethyl ether acetate. $CH_3COOC_2H_4OC_2H_4OCH_3$. Mol.wt. 162.18. A colorless, water-soluble liquid. Sp.gr. 1.0396; b.p. 209.1° C.; flash point, 180° F. *Use:* As a solvent and plasticizer for cellulose ester lacquers, printing inks, etc.
Carbide & Carbon Chemicals Corp.

**Methyl Carbitol Formal.** $CH_2(CH_3OCH_2CH_2OCH_2CH_2O)_2$. Mol.wt. 252.30. A liquid with a b.p. of 305° C.; sp.gr. of 1.0402; freezing point of —37.4° C.; and a flash point of 310° F. *Use:* As a plasticizer for resins, especially those intended for low temperature use, and where low hydrocarbon solubility is desired.
Carbide & Carbon Chemicals Corp.

**Methyl Cellosolve.** Ethylene glycol monomethyl ether. $CH_3OCH_2CH_2OH$. Mol.wt. 76.09. A stable, colorless liquid with a mild odor. Sp.gr. 0.9664; b.p. 124.2° C.; freezing point, —89.5° C.; flash point, 115° F.; completely soluble in water; miscible with most organic solvents. *Use:* As a solvent for cellulose esters, resins, enamels, varnishes, etc.
Carbide & Carbon Chemicals Corp.

**Methyl Cellosolve Acetal.** $CH_3CH(OCH_2CH_2OCH_3)_2$. Mol.wt. 178.22. A liquid with a b.p. of 207.0° C.; sp.gr. of 0.9762; and flash point of 205° F. It is completely soluble in water. *Uses:* As a solvent under neutral or slightly alkaline conditions; and as a source of acetaldehyde by hydrolysis in the presence of acids.
Carbide & Carbon Chemicals Corp.

**Methyl Cellosolve Acetate.** Ethylene glycol monomethyl ether acetate. $CH_3COOCH_2CH_2OCH_3$. Mol.wt. 118.13. A stable, colorless liquid with a mild characteristic odor. Sp.gr. 1.0067; b.p. 144.5°C.; freezing point, —65.1° C.; flash point, 140° F.; completely soluble in water; miscible with most organic solvents. *Use:* As a solvent for cellulose nitrate, cellulose acetate, gums, resins, waxes, oils, lacquers, etc.
Carbide & Carbon Chemicals Corp.

**Methyl Cellosolve Formal.** $CH_2(OCH_2CH_2OCH_3)_2$. Mol.wt. 164.19. A colorless, liquid ether, completely miscible with water. B.p. 201.0° C.; sp.gr. 0.999. *Use:* As a general solvent.
Carbide & Carbon Chemicals Corp.

**Methyl Chloride A.** A refrigerant consisting of 99% methyl chloride and 1% acrolein, which acts as a warning agent. A colorless, stable, gas at ordinary temperatures (b.p. —23.9° C.); slightly soluble in water; soluble in most organic solvents; moderately flammable.
DuPont Co.

**Methyl Chloride N.** A refrigerant consisting of 99% methyl chloride and 0.3% methyl nitrite, which acts as a leak detector. Its properties are the same as for Methyl Chloride A.
DuPont Co.

**Mothex.** A DDT insecticidal spray designed especially for the control of moths. It consists of a solution of DDT in a nonflammable, nonstaining, rapid-evaporating solvent mixture that is noninjurious to fabrics.

**Nacconol EP.** Sodium alkyl aryl sulfonate. Dry, cream-colored flakes with a slight odor and bitter flavor. Soluble in water—3% at room temperature—and in common organic solvents; and stable in hard water, and boiling solutions of $H_2SO_4$ and NaOH. *Use:* As an antipitting agent in nickel plating.
National Aniline Div. Allied Dye & Chemical Corp.

**Nacconol HG.** An alkyl aryl sulfonate in the form of light cream-colored flakes having an organic content of 55%. It possesses good stability in acid and alkaline solutions. *Use:* As a surface-active agent in the manufacture of salt-water, all-purpose soaps.
National Aniline Div. Allied Chemical & Dye Corp.

**Naphthol Mineral Spirits (Amsco).** A water-white, noncorrosive, petroleum naphtha solvent. Initial b.p. 290° F.; sp.gr. 0.7669. *Use:* as a general solvent.
AMERICAN MINERAL SPIRITS CO.

**Naphtholite (Amsco).** A water-white, noncorrosive petroleum naphtha solvent. Initial b.p. 205-210° F.; sp.gr. 0.74-0.76. *Use:* As a general solvent.
AMERICAN MINERAL SPIRITS CO.

**Naphtholite Special (Amsco).** A water-white, noncorrosive petroleum naphtha solvent. Initial b.p. 215-230° F.; sp.gr. 0.73-0.75.
AMERICAN MINERAL SPIRITS CO.

**Nevasota.** A refined creosote oil designed for wood preservation by brush, spray, or dip processes. Its high toxicity and powerful permanent action give low-cost protection against wood decay for many years. *Use:* For rot-proofing of barges, barns, boats, chicken coops, farm buildings, railroad ties, wood shingles, telephone poles, warehouses, etc. NEVILLE CO.

**Para Baco.** Para-dichlorobenzene. $C_6H_4Cl_2$. Mol.wt. 147.01. Clear, white crystals that melt at 127.4° F. Sp.gr. 1.53; insoluble in water; soluble in chloroform, benzene, carbon tetrachloride, acetone, and gasoline. *Uses:* As a mothproofing agent; in insecticidal and germicidal compositions; for controlling blue mold on tobacco plants; as a deodorant; and in the manufacture of dyestuffs and intermediates. SOLVAY PROCESS CO.

**Paradow.** Para-dichlorobenzene. $C_6H_4Cl_2$. Mol.wt. 147.01. Volatile, white crystals with a penetrating odor. Sp.gr. 1.27; m.p. 53° C.; insoluble in water; soluble in alcohol, benzene, and ether. *Use:* In the preparation of moth-proofing compositions, insecticides, germicides, etc. DOW CHEMICAL CO.

**Parapont.** Para-dichlorobenzene. $C_6H_4Cl_2$. Mol.wt. 147.01. A colorless, crystalline solid with an aromatic odor. It is soluble in alcohol, benzene, carbon disulfide, and ether; m.p. about 53° C. *Uses:* In the manufacture of deodorant blocks and sanitary specialties, insecticidal and germicidal compositions; as a moth-proofing agent; and as a soil fumigant. DUPONT CO.

**Para Scalecide.** A combination of para-dichlorobenzene and SCALECIDE (a miscible oil containing growth-promoting hormones) for controlling boring insects on fruit and shade trees. B. G. PRATT CO.

**Pariflux.** A special grade of calcium fluoride for welding use. It contains a minimum of 95% $CaF_2$; it is ground so that 100% passes through a 40-mesh screen; and it is available in two particle-size ranges. GRADE I is sized as follows: 20-30% on 100 mesh, 25-30% on 200, and 45-50% through 200. GRADE P is sized: 0-5% on 60 mesh, 5-15% on 100, and 50-60% through 200. FOOTE MINERAL CO.

**Perm-A-Chlor.** A highly stabilized, trichlorethylene-base degreasing solvent that is used to remove all types of oils, greases, and waxes from metal surfaces.
DETREX CORP.

**Perm-A-Kleen.** A perchlorethylene-base dry-cleaning solvent that is suitable for the cleaning of all types of clothing. DETREX CORP.

**Per-Clene.** Perchlorethylene. $C_2Cl_4$. Mol.wt. 165.85. A colorless, liquid, chlorinated hydrocarbon with an etherlike odor. It is nonflammable, nonexplosive, and stable in the presence of moisture. Sp.gr. 1.63; b.p. 120.8° C. *Uses:* As a metal-degreasing and dry-cleaning solvent; in chemical synthesis; as a fungicide; as an extraction agent; and as an anthelminthic remedy.
DUPONT CO.

**Perchloron.** A free-flowing, high-strength calcium hypochlorite with an available chlorine content of not less than 70%. *Use:* For operations requiring chlorine, such as sterilization, oxidation, odor control, algae control, etc.
PENNSYLVANIA SALT MFG. CO.

**Permatox A.** A 5% solution of pentachlorophenol (DOWICIDE 7) in petroleum solvents. *Use:* For the preservative treatment of lumber, poles, posts, and other forest products.
A. D. CHAPMAN & CO.

**Permatox A Concentrate.** A dark-colored, oily liquid consisting of a 41% solution of pentachlorphenol (DOWICIDE 7) in blended petroleum solvents. Weight, 9.9 lb./gal. at 100° F.; flash point, above 100° F. One part of PERMATOX A CONCENTRATE is diluted with 10 parts by volume of a petroleum solvent, weighing approx. 7 lb./gal., to make a standard solution of PERMATOX A, which contains 5% pentachlorophenol by weight.
A. D. CHAPMAN & CO.

**Phosflake.** A uniform blend of caustic soda (NaOH) and trisodium phosphate ($Na_3PO_4$) prepared in flake form. It is a detergent, especially suitable for machine bottle washing.
COLUMBIA CHEMICAL DIV., PITTSBURGH PLATE GLASS CO.

**Ponsolve.** A colorless solution with a mild, spirituous odor and possessing high solvent power. It contains 100 parts of a specially denatured alcohol, 5 parts ethyl acetate, and 1 part aviation-grade gasoline. *Uses:* As a solvent for shellac, gums, resins, stains, and dyes; as a constituent of paint and varnish removers; as a component of natural and artificial leather coatings; and as a solvent for cleaning compounds, insecticides, polishes, etc.
DUPONT CO.

**Santochlor.** Para-dichlorobenzene. $C_6H_4Cl_2$. Mol.wt. 147.01. White crystals; m.p. 52.8° C. (min.). *Uses:* As an intermediate in the manufacture of dyestuffs; as a deodorant, mothicide, and larvacide; and in the control of peach tree borers.
MONSANTO CHEMICAL CO.

**Snowflake.** A white, crystalline powder soldering flux for hand-soldering operations. It is a mixture of zinc chloride and ammonium chloride.
DUPONT CO.

**Solvenol.** A complex combination of monocyclic terpene hydrocarbons. *Uses:* As a solvent in varnishes and enamels, especially for finishes that are force-dried or baked, such as wire enamels; and as a solvent in rubber reclaiming.
HERCULES POWDER CO.

**Sovasol.** A series of petroleum solvents manufactured in two grades. The first grade (SOVASOLS 1, 2, 3, 4, 5, and 6) are straight-run naphthas of varying boiling ranges, and the second grade (SOVASOLS 73, 74, and 75) are catalytically processed, high-solvent-power naphthas. Some of the properties of these solvents are as follows:

| SOVASOL NO. | SP.GR. | DISTILLATION RANGE, ° F. |
|---|---|---|
| 1 | 0.7089 | 115-254 |
| 2 | 0.7005 | 125-217 |
| 3 | 0.7401 | 200-271 |
| 4 | 0.7632 | 242-318 |
| 5 | 0.7865 | 304-385 |
| 6 | 0.8095 | 368-460 |
| 73 | 0.7839 | 195-285 |
| 74 | 0.8443 | 278-360 |
| 75 | 0.8315 | 316-395 |

SOCONY-VACUUM OIL CO.

**Styramic.** A polystyrene chlorinated diphenyl compound used as a thermoplastic injection and extrusion molding material. It combines excellent electrical properties with superior heat resistance which, however, is not as high as that of STYRANIC HT or CEREX. *Use:* Principally for high-frequency insulation purposes. MONSANTO CHEMICAL CO.

**Sulchlor 36M.** A sulfurized and chlorinated mineral oil containing 3½% sulfur and ¾% chlorine. *Use:* In the manufacture of cutting oils and drawing and broaching compounds. CARLISLE CHEMICAL WORKS

**Sulchlor 66.** An odorless, sulfurized, and chlorinated sperm oil base for the manufacture of cutting oils and drawing compounds. It contains a minimum of 8% sulfur and 8% chlorine. It is stable at high temperatures and possesses high antiwelding properties and good film strength. CARLISLE CHEMICAL WORKS

**Synasol.** A water-white liquid solvent composed of 100 parts denatured ethyl alcohol, 5 parts ethyl acetate, and 1 part aviation gasoline. It possesses a mild agreeable odor. Sp.gr. 0.816; b.p. 70-80° C.; flash point, 70° F.; weight, 6.81 lb./gal. *Uses:* As a solvent for resins, stains, dyes, greases, etc.; in the manufacture of varnish removers, artificial leather, paper and fabric coatings, disinfectants and insecticides, and soldering fluxes; in oil refining; in polishing and cleaning compounds, etc. CARBIDE & CARBON CHEMICALS CORP.

**Trek.** An antifreeze liquid containing methanol and rust inhibitors and dyed a deep purple color. Sp.gr. 0.8007; water content, not over 0.1%; methanol content, not less than 99.5%. *Use:* As an antifreeze for automotive cooling systems. EVEREADY DIV. NATIONAL CARBON CO.

**Tri-Clene.** Trichlorethylene. $C_2HCl_3$. Mol.wt. 131.40. A colorless chlorinated hydrocarbon with a characteristic ethereal odor. It is nonflammable, practically nonexplosive, and has low latent and specific heats. Sp.gr. 1.472; b.p. 86.7° C.; practically insoluble in water. *Uses:* As a solvent for dry cleaning and spotting of textiles; as a metal-degreasing solvent; and as a solvent for animal and vegetable oil extraction. DUPONT CO.

**Triasol.** A mixture of 70% trichloroethylene by volume, and 30% ethylene dichloride. A water-white, nonflammable liquid; boiling range, 80-85° C.; sp.gr. 1.334-1.339. *Uses:* As a solvent for oils, waxes, and resins; and as a metal and textile cleaner. CARBIDE & CARBON CHEMICALS CORP.

**V-Meth-L.** Methyl chloride of refrigeration grade. $CH_3Cl$. Mol.wt. 50.481. A colorless liquid with a pleasant odor. B.p. —10.66° F.; critical temp. 289.6° F.; critical pressure, 969.2 lb./sq.in., abs. VIRGINIA SMELTING CO.

**Vatrolite.** A concentrated solution of sodium hydrosulfite. $Na_2S_2O_4$. Mol.wt. 174.11. *Uses:* As a reducing agent in the vat dyeing of textiles and in fast-color dyeing; for bleaching soap; and for removing colors from textile fabrics. ROYCE CHEMICAL CO.

**Vatsol OS.** The sodium salt of an alkyl naphthalene sulfonic acid. It is a powdered wetting agent. *Uses:* With insecticide dusts to promote the spreading of the insecticide; and with hydrochloric acid to remove poison residue from fruit. PEARSON-FERGUSON CHEMICAL CO.

**Vatsol OT.** A higher alcohol ester of sodium sulfosuccinic acid. It is a powdered wetting agent used to promote the spreading of insecticidal dusts and sprays. PEARSON-FERGUSON CHEMICAL CO.

### ORGANIC SOLVENTS USED FOR CLEANING OR DEGREASING OPERATIONS

(Brandt, McConnell and Flinn: Public Health Reports, Vol. 61, No. 5, pp. 132-143, reprint No. 2691.)

| Code No. | Name or trade name of product | Composition (per cent) | | | | | Boiling range (° C.) | Supplier or manufacturer |
|---|---|---|---|---|---|---|---|---|
| | | Aromatics (mac. 50–200 p. p. m.) | Halogenated hydrocarbons (mac. 50–200 p. p. m.) | Alcohols, esters, ethers, and ketones (mac. 200–400 p. p. m.) | Paraffins and naphthenes (mac. 500–1,000 p. p. m.) | Other | | |
| C- 1 | Actusol (grease solvent) | | | | 100 | | 155–205 | Warren Refining Co., Cleveland, Ohio. |
| C- 2 | Amercoat #10 | 29 | | 58 | 13 | | 110–158 | Gregg Co., Philadelphia, Pa. |
| C- 3 | Amercoat #12 | 27 | | 64 | 9 | | 58–164 | Do. |
| C- 4 | APCO–140 | | | | 100 | | 180–220 | Bertram Naphtha Co., Kansas City, Mo. |
| C- 5 | Ber-to-sol | 5 | 60 | | 35 | | 84–144 | Do. |
| C- 6 | Blankrola | 9 | 45 | | 46 | | 128–175 | Addressograph-Multigraph Corp., Cleveland, Ohio. |
| C- 7 | Casite Sludge Solvent | 45 | | | 55 | | 170–260 | Graff Motor Co., Rapid City, S. D. |
| C- 8 | CC Sol | 10 | | 17 | 73 | | 80–146 | Bertram Naphtha Co., Kansas City, Mo. |
| C- 9 | Cellosolve Acetate | | | 100 | | | 155–158 | Carbide & Carbon Chemicals Corp., Detroit, Mich. |
| C-10 | Cleaners Naphtha | | | | 100 | | 155–220 | |
| C-11 | Cleaning Fluid | | | | 100 | | 145–200 | Quartermaster, Aberdeen Proving Ground, Aberdeen, Md. |
| C-12 | Colonel Speedy Carbon and Rust Remover. | | | 45 | | Water | 70–100 | Jersey City Quartermaster Depot, Jersey City, N. J. |
| C-13 | Dearboline Cleaning Compound | | | | 100 | | 185–300 | Dearborn Chemical Co., Philadelphia, Pa. |
| C-14 | Degreasal | | 100 | | | | 70–110 | General Electric Supply Co., Denver, Colo. |
| C-15 | Degreasal Hi T | | | | 100 | | 160–222 | General Electric Supply Co., Evansville, Ind. |
| C-16 | Ditto Fluid | | | 100 | | | 78 app. | Ditto Inc., Chicago, Ill. |
| C-17 | Dry Cleaners Solvent | | | | 100 | | 156–196 | |
| C-18 | Dry Cleaning Fluid | | 25 | | 75 | | 75–140 | Shell Oil Co., Baltimore, Md. |
| C-19 | Dry Cleaning Solvent | | | | 100 | | 160–205 | Paris Oil Co., Paris, Tex. |
| C-20 | Dry Cleaning Solvent (PS 661A) | | | | 100 | | 145–210 | Standard Oil Co. (Ohio), Cleveland, Ohio |
| C-21 | Finger Print Remover | | | | 100 | | | |
| C-22 | Gas-O-Clens | | | | 100 | | 80–150 | Gulf Oil Corp., Augusta, Ga. |
| C-23 | Hylite | | | | 100 | | 55–175 | Socony Vacuum Oil Co., New York, N. Y. |
| C-24 | Imperial Washer Cleaner | 20 | 70 | | 10 | | 75–120 | Martindale Electric Co., Cleveland, Ohio |
| C-25 | Ink Cleaner #77 | | | 80 | 20 | | 75–235 | Superior Type Co., Chicago, Ill. |
| C-26 | Keelite Z. C. Stripper | 25 | | 65 | 10 | | 60–135 | Keelite Products Inc., Houston, Tex. |
| C-27 | Kerosene | | | | 100 | | | |
| C-28 | Kleen-O-Type | 35 | 65 | | | | 75–80 | Gulf States Sales Co., Birmingham, Ala. |
| C-29 | Lacquer Thinner #CT-350 | 16 | | 47 | 37 | | 78–134 | E. I. duPont, Wilmington, Del. |
| C-30 | Lithotine | | | | 100 | | 150–210 | Sinclair and Valentine, Baltimore, Md. |
| C-31 | Loosite | 30 | | 8 | 62 | | 60–270 | Petroleum Solvents Corp., New York, N. Y. |
| C-32 | Magnolia Sovalsol | | | | 100 | | 160–200 | Magnolia Petroleum Co., Amarillo, Tex. |
| C-33 | Magnus Carbon Remover | | 55 | | | Cresylic acid | | |
| C-34 | Magnusol | | | | 100 | | 130–190 | Magnus Chemical Co., Garwood, N. J. |
| C-35 | Mineral Spirits | | | | 100 | | 150–200 | Standard Oil Co., Talladega, Ala. |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| C-36 | Mineral Spirits #12 | | | | 100 | | 160-210 | Anderson Prichard Oil Co., Newark, N. J. |
| C-37 | Naphtha, Cleaners | | | | 100 | | 155-220 | |
| C-38 | Naphtha, V M & P | 11 | | | 89 | | 112-141 | Jamison Oil Co., Omaha, Nebr. |
| C-39 | Nu-film #22 | 10 | | 70 | 20 | | 65-140 | Cook Paint and Varnish Co., Omaha, Nebr. |
| C-40 | O'Brien Lacquer Thinner L-156-XX | 3 | | 40 | 57 | | | O'Brien Varnish Co., South Bend, Ind. |
| C-41 | Paco Solvent (denatured alcohol) | | | 100 | | | 75-100 | Industrial Chemical Co., Omaha, Nebr. |
| C-42 | Paint and Varnish Remover | 50 | | 50 | | | 64-75 | V. J. Dolman and Co., Chicago, Ill. |
| C-43 | Penotrite | | 20 | | 80 | | 139-186 | General Solvents, Rochester, N. Y. |
| C-44 | Ponsolve | | | 100 | | | 73-79 | E. I. duPont, Wilmington, Del. |
| C-45 | Rifle Bore Cleaner | | | | 80 | Water | 76-260 | S. C. Johnson & Son, Inc., Racine, Wis. |
| C-46 | Shamrock Solvent | | | | 100 | | 160-200 | Shamrock Oil & Gas, Amarillo, Tex. |
| C-47 | Shellacol | | | 100 | | | 78 app. | Shell Oil Co., New York, N. Y. |
| C-48 | Solvasol #5 (Varnoline) | | | | 100 | | 160-190 | Socony Vacuum Oil Co., New York, N. Y. |
| C-49 | Solvental | | | | 94 | Soap | 185-245 | Solvental Chemical Products Co., Pittsburgh, Pa. |
| C-50 | Sovalsol, Magnolia | | | | 100 | | 160-200 | Magnolia Petroleum Co., Amarillo, Tex. |
| C-51 | Stanisol Solvent Mixture | | | | 100 | | 150-200 | Standard Oil Co., New York, N. Y. |
| C-52 | Stoddard Solvent | | | | 100 | | 157-188 | Phillips Petroleum Co., St. Louis, Mo. |
| C-53 | Stoddard Solvent | | | | 100 | | 150-200 | W. J. Hough Co., Chicago, Ill. |
| C-54 | Stoddard Solvent | | | | 100 | | 145-195 | Aetna Oil Co., Louisville, Ky. |
| C-55 | Synosol | | | 100 | | | 84 app. | Cook Paint & Varnish Co., Chicago, Ill. |
| C-56 | Testors Thinner | | | 60 | 40 | | 60-110 | Testor Chemical Co., Rockford, Ill. |
| C-57 | Tomac Thinner | | | | 100 | | 60-210 | American Hospital Supply Co., Chicago, Ill. |
| C-58 | Tri Sol | | 45 | | 55 | | 82-209 | Bertram Naphtha Co., Kansas City, Mo. |
| C-59 | Type Wash | 39 | | 38 | 23 | | 64-142 | Barco Chemical Products Co., Chicago, Ill. |
| C-60 | Varnish and Paint Remover | 50 | | 50 | | | 64-75 | V. J. Dolman & Co., Chicago, Ill. |
| C-61 | Varsol | | | | 100 | | 150-200 | Standard Oil Co., New York, N. Y. |
| C-62 | V M and P Naphtha | 11 | | | 89 | | 112-141 | Jamison Oil Co., Omaha, Nebr. |
| C-63 | Washurite | | 25 | 5 | 70 | | 61-170 | Davidson Mfg. Co., Chicago, Ill. |

### ORGANIC SOLVENTS USED AS THINNERS

(Brandt, McConnell and Flinn: Public Health Reports, Vol. 61, No. 5, pp. 132-143, reprint No. 2691.)

| Code No. | Name or trade name of product | Composition (percent) | | | Boiling range (° C.) | Supplier or manufacturer |
|---|---|---|---|---|---|---|
| | | Aromatics (mac. 50–200 p. p. m.) | Alcohols, esters, ethers, and ketones (mac. 200–400 p. p. m.) | Paraffins and naphthenes (mac. 500–1,000 p. p. m.) | | |
| T- 1 | Acme Paint Thinner | 25 | 60 | 15 | 70–125 | Pittsburgh Auto Equipment Co., Meadville, Pa. |
| T- 2 | AMI Thinner | | | 100 | 100–250 | |
| T- 3 | ANA Thinner (enamel) | | 100 | | 82–128 | Sewell Paint Co., Kansas City, Mo. |
| T- 4 | Apco Thinner | 14 | | 86 | 122–147 | Bertram Naphtha Co., Kansas City, Mo. |
| T- 5 | Bitumastic Thinner | | | 100 | 151–191 | McJunkin Supply Co., Charleston, W. Va. |
| T- 6 | Black Nitrocellulose Lacquer Enamel | 10 | 50 | 40 | 70–140 | Wabash Products Co., Terre Haute, Ind. |
| T- 7 | Black Stencil Paint Thinner | | | 100 | 158–195 | Phelan Faust, St. Louis, Mo. |
| T- 8 | Brit-Mark Stencil Ink Thinner | | 65 | 35 | 125–180 | Diagraph Bradley Stencil Machine Corp., St. Louis, Mo. |
| T- 9 | Butyl Cellosolve Thinner | | 100 | | 171 | Carbide and Carbon Chemical Corp., New York, N. Y. |
| T-10 | Carbitol | | 100 | | 198 | Do. |
| T-11 | Cellulose Nitrate Lacquer Thinner | | 60 | 40 | 70–125 | Pittsburgh Plate Glass Co., South Bend, Ind. |
| T-12 | Chocolate Brown Lacquer Thinner | 5 | 50 | 45 | 65–120 | Jones-Dabney, Louisville, Ky. |
| T-13 | Chromated Lacquer Thinner (Reducer #66) | 100 | | | | Grand Rapids Varnish Co., Grand Rapids, Mich. |
| T-14 | Duco #3614 | | 60 | 40 | 37–147 | E. I. duPont Co., Wilmington, Del. |
| T-15 | Duco Thinner (K–1486) | 29 | 42 | 29 | 64–142 | Arthur Fulmer, Memphis, Tenn. |
| T-16 | DuPont #200 Paint Thinner | 55 | | 45 | 187–226 | Koch's Paint & Supply Co., Burlington, Iowa |
| T-17 | DuPont #3322 Lacquer Thinner | 30 | 50 | 20 | 75–139 | E. I. duPont Co., Wilmington, Del. |
| T-18 | DuPont Lacquer Thinner #3673 | | 45 | 55 | 85–180 | Do. |
| T-19 | DuPont "Red" Thinner #3450 | | 70 | 30 | 60–120 | E. I. duPont Co., Dallas, Tex. |
| T-20 | DuPont Synthetic Reducer (T–8879) | | | 100 | 80–125 | The W. E. Wright Co., Akron, Ohio. |
| T-21 | DuPont Synthetic Reducer (T–8879) | 25 | | 75 | 136–204 | E. I. duPont Co., St. Louis, Mo. |
| T-22 | DuPont Thinner Mix | | 50 | 50 | | E. I. duPont Co., Wilmington, Del. |
| T-23 | Enamel Thinner | | | 100 | 90–120 | Sewell Paint & Varnish Co., Dallas, Tex. |
| T-24 | Ethyl Cellulose Lacquer Thinner | 15 | 50 | 35 | 78–143 | Sewell Paint & Varnish Co., Kansas City, Mo. |
| T-25 | Gensol | | Turpentine | | 145–175 | Newport Industries, Cincinnati, Ohio. |
| T-26 | Glyptal Thinner | 73 | | 27 | 126–143 | General Electric Supply Co., Pittsburgh, Pa. |
| T-27 | Hydro #1—Reducing—Spec. #2 | 75 | | 25 | 100–145 | Western Rosin & Turpentine Co., Detroit, Mich. |
| T-28 | Hydro Flex #195 Paint Thinner | 22 | | 78 | 118–187 | Phelan Faust Paint Mfg. Co., St. Louis, Mo. |
| T-29 | Ink Paste Thinner | | | 100 | 165–201 | Howard Flint Ink Co., Houston, Tex. |
| T-30 | Ink Reducer | 1 | | 99 | 153–205 | Howard Flint Ink Co., Detroit, Mich. |
| T-31 | Ink Thinner #77 | | 100 | | 77–97 | The Superior Type Co., Chicago, Ill. |
| T-32 | Insulating Varnish Thinner #5052 | 97 | | 3 | 79–126 | Westinghouse Electric Supply Co., Pittsburgh, Pa. |
| T-33 | Japan Dryer | | | 100 | 150–210 | |
| T-34 | Lacquer Enamel Thinner | 35 | 40 | 25 | 70–135 | O'Brien Varnish Co., South Bend, Ind. |
| T-35 | Lacquer Thinner | 45 | 25 | 30 | | Do. |

| | | | | | | |
|---|---|---|---|---|---|---|
| T-36 | Lacquer Thinner | 20 | 50 | 30 | | Cook Paint & Varnish Co., Houston, Tex. |
| T-37 | Lacquer Thinner | 11 | 51 | 38 | 69–134 | Pittsburgh Plate Glass Co., St. Louis, Mo. |
| T-38 | Lacquer Thinner | 15 | 45 | 40 | 72–137 | Gilman Paint & Varnish Co., Chattanooga, Tenn. |
| T-39 | Lacquer Thinner 250-C-636 | 15 | 55 | 30 | 75–140 | Cook Paint & Varnish Co., Houston, Tex. |
| T-40 | Lacquer Thinner #1055A | 2 | 72 | 26 | 78–120 | Sewell Paint & Varnish Co., Kansas City, Mo. |
| T-41 | Lacquer Thinner #1173B | 20 | 52 | 28 | 79–141 | P. D. George Co., St. Louis, Mo. |
| T-42 | Lacquer Thinner (FL-19830) | 15 | 60 | 25 | 80–139 | Pittsburgh Plate Glass Co., St. Louis, Mo. |
| T-43 | Lacquer Thinner (FL-20300) | 5 | 60 | 35 | 72–135 | Pittsburgh Plate Glass Co., Birmingham, Ala. |
| T-44 | Lacquer Thinner (U. L. 10045) | 15 | 60 | 25 | 70–140 | O'Brien Varnish Co., South Bend, Ind. |
| T-45 | Mineral Spirits | | | 100 | 156–198 | Standard Oil Co., Huntsville, Ala. |
| T-46 | Naphtha | 5 | | 95 | 88–150 | |
| T-47 | Naphtha | 10 | | 90 | 96–143 | Jamieson Oil Co., Omaha, Nebr. |
| T-48 | Nitrocellulose Lacquer Thinner | 45 | 35 | 20 | 75–140 | F. J. Donahoe Varnish Co., Detroit, Mich. |
| T-49 | Nitrocellulose Nitrate Dope Thinner | 30 | 55 | 15 | 75–140 | Grand Rapids Varnish Co., Grand Rapids, Mich. |
| T-50 | N. R. C. Thinner | 3 | 70 | 27 | 67–121 | E. I. duPont Co., Wilmington, Del. |
| T-51 | O'Brien Lacquer Thinner (L-165-3) | 46 | 30 | 24 | | O'Brien Varnish Co., South Bend, Ind. |
| T-52 | Paint #29232A Thinner | | | 100 | 98–136 | Pittsburgh Plate Glass Co., Cleveland, Ohio. |
| T-53 | Pentol O. D. Enamel Thinner | 20 | 10 | 70 | | Sewell Paint & Varnish Co., Kansas City, Mo. |
| T-54 | Phenolic Thinner | | 100 | | | Stoner & Nudge Co., Pittsburgh, Pa. |
| T-55 | Pittsburgh Minimax | | 36 | 64 | | Pittsburgh Plate Glass Co., Pittsburgh, Pa. |
| T-56 | Pyroxyline Thinner | 4 | 55 | 41 | | S. C. Johnson & Son, Inc., Racine, Wis. |
| T-57 | Red Lacquer Thinner | 15 | 50 | 35 | 68–145 | Pittsburgh Plate Glass Co., Memphis, Tenn. |
| T-58 | Reducer #74 | | | 100 | 145–205 | Sherwin-Williams Paint Co., Baltimore, Md. |
| T-59 | Sea Lac Lacquer Thinner | 30 | 40 | 30 | 65–130 | Duralac Chemical Corporation, Newark, N. J. |
| T-60 | Sewell #3551 Paint Thinner | 75 | | 25 | 134–175 | Sewell Paint & Varnish Co., Kansas City, Mo. |
| T-61 | Sherwin-Williams Paint Thinner | 5 | | 95 | 64–130 | Sherwin-Williams Co., Dallas, Tex. |
| T-62 | Sol Naphtha | | | 100 | 160–194 | Shell Petroleum Co., New York, N. Y. |
| T-63 | Solox | | 100 | | 77–107 | McKesson-Robbins Co., Omaha, Nebr. |
| T-64 | Solvesso #1 Thinner | 60 | | 40 | 85–135 | Standard Oil Co., Norfolk, Va. |
| T-65 | Solvesso #3 | 70 | | 30 | 170–220 | Colonial Beacon Oil Co., Springfield, Mass. |
| T-66 | Solvesso—Sohio #1 | 70 | | 30 | 90–140 | Standard Oil Co., Akron, Ohio. |
| T-67 | Special Fast Dry Stencil Thinner | 18 | | 82 | 111–141 | Wm. H. Witte & Sons Paint Co., Burlington, Iowa. |
| T-68 | Spruance Thinner | 7 | 55 | 38 | 68–140 | Gilbert Spruance Co., Philadelphia, Pa. |
| T-69 | Stencil Thinner Solvent #50 | 25 | 65 | 10 | 50–195 | Garvey Fountain Brush & Ink Co., St. Louis, Mo. |
| T-70 | Superior #77 Ink Thinner | | 100 | | 80–200 | The Superior Type Co., Chicago, Ill. |
| T-71 | Synthetic Enamel Thinner | 40 | | 60 | 135–200 | |
| T-72 | Synthetic Thinner for O. D. Paint | | | 100 | 90–145 | Rinshed Mason Co., Detroit, Mich. |
| T-73 | Synthetic Thinner—Latex | | 60 | 40 | 70–165 | Dyke Motor Supply Co., Akron, Ohio. |
| T-74 | Thinner #30 | 60 | 40 | | 56–125 | Graybar Electric Co., Newark, N. J. |
| T-75 | Thinner #74 | | | 100 | 160–210 | Sherwin-Williams Co., Des Moines, Iowa. |
| T-76 | Thinner, Acid Proof Black Paint | 15 | | 85 | 120–147 | Testor Chemical Co., Rockford, Ill. |
| T-77 | Thinner, Cooks | 20 | 50 | 30 | | Cook Paint & Varnish Co., Houston, Tex. |
| T-78 | Thinner, G. E. #1500 | 70 | | 30 | 105–145 | Graybar Electric Co., Newark, N. J. |
| T-79 | Thinner, Lacquer #20 | 22 | 52 | 26 | 69–153 | Acme White Lead & Color Works, Detroit, Mich. |
| T-80 | Thinner, Lacquer (du Pont) (3-162-1) | 21 | 32 | 47 | | E. I. duPont, Co., Wilmington, Del. |
| T-81 | Thinner, Lacquer Enamel (3-162-A) | 20 | 50 | 30 | 70–130 | Wabash Products Co., Terre Haute, Ind. |
| T-82 | Thinner, Lacquer Enamel (Cooks) | 34 | | 66 | 67–141 | Cook Paint & Varnish Co., Kansas City, Mo. |
| T-83 | Thinner, Lacquer Enamel (Nitro Cellulose) | | 60 | 40 | | S. C. Johnson & Son., Inc., Racine, Wis. |
| T-84 | Thinner, Marking Ink #4 | | 100 | | 124 | Hilton Hawley Co., Cincinnati, Ohio. |
| T-85 | Thinner—Paint (#76) | 10 | 65 | 25 | | |

*(Continued on next page)*

### Organic Solvents Used as Thinners—Cont'd

| Code No. | Name or trade name of product | Composition (percent) | | | Boiling range (° C.) | Supplier or manufacturer |
|---|---|---|---|---|---|---|
| | | Aromatics (mac. 50–200 p. p. m.) | Alcohols, esters, ethers, and ketones (mac. 200–400 p. p. m.) | Paraffins and naphthenes (mac. 500–1,000 p. p. m.) | | |
| T–86 | Thinner, Pedigree #150 | 100 | | | 133–151 | H. A. Holden, Inc., Minneapolis, Minn. |
| T–87 | Thinner, Testor's | 26 | 33 | 41 | | Testor Chemical Co., Rockford, Ill. |
| T–88 | Thinner, Witte's Lacquer Thinner | 26 | 30 | 44 | 82–124 | Wm. H. Witte & Sons Paint Co., Burlington, Iowa. |
| T–89 | V. M. & P. Naphtha | 5 | | 95 | 98–176 | Socony Vacuum Co., St. Louis, Mo. |
| T–90 | Warren's Deodorized Leptine | | | 100 | 165–210 | Warren Paint and Color Co., Nashville, Tenn. |
| T–91 | Wash #5816 Thinner | 10 | 75 | 15 | 70–140 | George Rutledge Co., Montclair, N. J. |
| T–92 | Westinghouse #8110 Thinner | | 100 | | 73–107 | Westinghouse Electric Supply Co., Pittsburgh, Pa. |
| T–93 | Westinghouse Tuffernell Thinner 1609 | 3 | | 97 | 155–196 | Westinghouse Electric Supply Co., St. Paul, Minn. |
| T–94 | Xylol Solvesso | 100 | | | 135–180 | Standard Oil Co., Akron, Ohio. |
| T–95 | Zapon Cotite A | 15 | 80 | 5 | 70–130 | Zapon Division, Atlas Powder Co., Philadelphia, Pa. |

## Industrial Safety Rules and Regulations

The following tables are those adopted by the Division of Industrial Safety of the State of California. With few exceptions these are the same as those in use by other states of the nation. From time to time experience reveals that certain of these allowable concentrations must be changed:

### SUGGESTED MAXIMUM PERMISSIBLE CONCENTRATIONS
(Toxic Thresholds)

| *Substance* | *Unit of Measurement* | *Limit* |
|---|---|---|
| **Dusts:** | Million particles | |
| Total Dust (0.5 to 10.0 microns) | per cubic foot (except as specified<br>“ below)<br>“ | 50 |
| **Mineral Dusts** (0.5 to 10.0 microns): | “ | |
| Asbestos | “ | 5 |
| Cement | “ | 15 |
| Silica (less than 10%) | “ | 50 |
| Silica (10% to 50%) | “ | 10 |
| Silica (more than 50%) | “ | 5 |
| Slate | “ | 15 |
| Talc | “ | 15 |
| **Metallic Dusts and Fumes:** | | |
| | Milligrams | |
| Cadmium | per cubic meter | 0.1 |
| Chromium (acid, chromate or | “ | 0.1 |
| dichromate) | “ | 0.15 |
| Lead and lead compounds | “ | 6.0 |
| Manganese | “ | 0.1 |
| Mercury | “ | 30.0 |
| Total metal fumes | “ | 15.0 |
| Zinc oxide fumes | | |
| **Miscellaneous Dusts and Fumes:** | | |
| Chlorides (fumes) | Parts per million | 15.0 |
| Chlorinated diphenyls | “ | 1.0 |
| Chlorinated Naphthalenes (except Trichloronaphthalene) | “ | 1.0 |
| Fluorides (dusts and fumes) | “ | 2.0 |
| Tetryl, T.N.T. and D.N.T. | “ | 1.5 |
| Triehloronaphthalene | “ | 10.0 |
| **Gases and Vapors:** | | |
| Acetate, amyl | Parts per million | 400 |
| Acetate, butyl | “ | 400 |
| Acetate, ethyl | “ | 400 |
| Acetate, methyl | “ | 400 |
| Acetate, propyl | “ | 400 |
| Acetone (see Ketones) | | |
| Acetylene | Use explosive limit | |
| Acids | | |
| Hydrochloric acid | Parts per million | 10 |
| Hydrogen cyanide | “ | 20 |
| Hydrogen fluoride | “ | 3 |
| Nitric acid | “ | 10 |
| Sulfuric acid | “ | 10 |
| Acrolein (see Aldehydes) | | |
| Acrylonitrile | “ | 20 |
| Alcohol, amyl | “ | 200 |
| Alcohol, butyl | “ | 200 |
| Alcohol, ethyl | “ | 1000 |
| Alcohol, methyl | “ | 200 |

| *Substance* | *Unit of Measurement* | *Limit* |
|---|---|---|
| Aldehydes, total | Parts per million | 10 |
| Acrolein | " | 1 |
| Formaldehyde | " | 10 |
| Ammonia | " | 100 |
| Aniline | " | 5 |
| Arsine | " | 1 |
| Benzol | " | 100 |
| Bromide, methyl | " | 30 |
| Bromine | " | 1 |
| Carbon dioxide | " | 5000 |
| Carbon disulfide | " | 20 |
| Carbon monoxide | " | 100 |
| Carbon tetrachloride | " | 100 |
| "Cellosolve" (see Glycols) | | |
| Chlorine | " | 1 |
| Chloroform | " | 100 |
| Cyanogen and compounds | " | 20 |
| Dichlorobenzene | " | 75 |
| Dichloroethylene | " | 200 |
| Dichloromethane | " | 200 |
| Dimethylaniline | " | 5 |
| Ether, ethyl | " | 400 |
| Ethylene dichloride (dichloroethane) | " | 100 |
| Ethylene oxide | " | 250 |
| Formaldehyde (see Aldehydes) | | |
| Gasoline | " | 1000 |
| Glycols | | |
| Diethylene glycol monoethyl ether ("carbitol") | " | 50 |
| Ethylene glycol monobutyl ether (butyl "cellosolve") | " | 50 |
| Ethylene glycol monoethyl ether ("cellosolve") | " | 500 |
| Ethylene glycol monomethyl ether acetate ("cellosolve" acetate) | " | 50 |
| Ethylene glycol monomethyl ether (methyl "cellosolve") | " | 50 |
| Hydrogen cyanide (see Acid) | | |
| Hydrogen fluoride (see Acid) | | |
| Hydrogen sulfide | " | 20 |
| Ketone, dimethyl (acetone) | " | 500 |
| Ketone, methyl butyl (hexanone) | " | 500 |
| Ketone, methyl ethyl (butanone) | " | 500 |
| Ketone, methyl isobutyl (hexone) | " | 500 |
| Ketone, methyl propyl (pentanone) | " | 500 |
| Monochlorobenzene | " | 75 |
| Nitrobenzene | " | 5 |
| Nitrogen, oxides of | " | 25 |
| Ozone | " | 1 |
| Phosgene | " | 1 |
| Phosphine | " | 1 |
| Phosphorus trichloride | " | 1 |
| Styrene monomer | " | 400 |
| Sulfur dioxide | " | 10 |
| Tetrachlorethane | " | 10 |
| Tetrachlorethylene (Perchlorethylene) | " | 200 |
| Toluol (Toluene) | " | 200 |
| Trichlorethylene | " | 200 |
| Turpentine | " | 200 |
| Vinyl cyanide (see Acrylonitrile) | | |
| Xylol (Xylene) | " | 200 |

## LOWER EXPLOSIVE LIMITS OF INFLAMMABLE VAPORS

| Gas or Vapor: | *Per cent by Vol.* | | *Per cent by Vol.* |
|---|---|---|---|
| Acetaldehyde | 4.0 | Ethylene oxide | 3.0 |
| Acetone | 2.2 | Furfural | 2.1 |
| Acetylene | 2.3 | Gasoline | 1.4 |
| Allyl alcohol | 3.0 | Heptane | 1.0 |
| Ammonia | 16.0 | Hexane | 1.2 |
| Amyl acetate | 1.1 | Hydrogen | 4.1 |
| Amyl alcohol | 1.2 | Hydrogen cyanide | 5.6 |
| Amyl chloride | 1.4 | Hydrogen sulfide | 4.3 |
| Amylene | 1.6 | Methane | 5.3 |
| Benzine | 1.2 | Methyl acetate | 4.1 |
| Benzol | 1.4 | Methyl alcohol | 6.0 |
| Benzyl chloride | 1.1 | Methyl bromide | 13.5 |
| Butadiene | 2.0 | Methyl chloride | 8.0 |
| Butane | 1.6 | Methyl butyl ketone | 1.2 |
| Butyl acetate | 1.7 | Methyl cyclohexane | 1.1 |
| Butyl alcohol | 1.7 | Methyl ethyl ether | 2.0 |
| Butylene | 1.7 | Methyl ethyl ketone | 1.8 |
| Carbon disulfide | 1.0 | Methyl formate | 5.0 |
| Carbon monoxide | 12.5 | Methyl propyl ketone | 1.5 |
| Cellosole | 2.6 | Naphthalene | 0.9 |
| Cellosolve, acetate | 1.7 | Nonane | 0.7 |
| Cyanogen | 6.6 | Octane | 0.8 |
| Cyclohexane | 1.3 | Pentane | 1.4 |
| Decane | 0.7 | Propane | 2.4 |
| Dichlorethylene | 9.7 | Propyl acetate | 2.0 |
| Diethylene oxide | 2.0 | Propyl alcohol | 2.5 |
| Ethane | 3.2 | Propylene | 2.0 |
| Ethyl acetate | 2.2 | Propylene dichloride | 3.4 |
| Ethyl alcohol | 3.3 | Pyridine | 1.8 |
| Ethyl bromide | 6.7 | Stoddard's solvent | 1.1 |
| Ethyl chloride | 3.6 | Styrene monomer | 1.1 |
| Ethyl ether | 1.7 | Toluene | 1.3 |
| Ethyl nitrate | 3.8 | Turpentine | 0.8 |
| Ethyl nitrite | 3.0 | Vinyl chloride | 4.0 |
| Ethylene | 3.0 | Xylene | 1.0 |
| Ethylene dichloride | 6.2 | | |

In addition to the table regarding toxic substances, each state has its own regulations as to the safe conduct of many industries. These are available to the industrial physician. Among these special Safety Rules and Safety Orders are the following:

Air Pressure Tank Safety Orders
Boiler Safety Orders
Compressed Air Safety Orders Governing Work in Compressed Air
Construction Safety Orders
Dusts, Fumes, Vapors and Gases Safety Orders
Electrical Safety Orders
Elevator Safety Orders
Engine Safety Orders

Gantry-Truck Safety Orders
General Safety Orders
Labeling of Injurious Substances Safety Orders
Laundry Safety Orders
Liquefied Petroleum Gases Safety Orders
Logging and Sawmill Safety Orders
Mechanical Power Transmission Safety Orders
Mine Safety Orders
Painting Safety Orders
Petroleum Industry Safety Orders
Pneumatic Explosives Loading Safety Orders
Quarry Safety Rules
Safety Rules for Gold Dredges
Ship and Boat Building Safety Orders
Steam Shovel and Locomotive Crane Safety Orders
Trench Construction Safety Orders
Tunnel Safety Rules
Window Cleaning Safety Orders
Women in Industry, Safety Orders for
Woodworking Safety Orders

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